

Tropical Abscess of the Liver.

TROPICAL ABSCESS OF THE LIVER.

*Thesis approved for the Degree of
Doctor of Medicine in the University of Madras,
1909.*

BY

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PREFACE

This work was originally undertaken as a thesis for the Degree of Doctor of Medicine and was successful in gaining commendation of the University of Madras, 1909. In this thesis which is now offered to the public I have made attempt to give an account of our present knowledge in the field of special pathology and treatment of Tropical Abscess of the Liver. This account, although imperfect, I have endeavoured to make as complete as possible in regard to all important and certainly established facts. I have been induced to publish it in the hope that it may prove of service to those interested in the pathology and treatment of the disease. The author is fully conscious of its many defects, to revise which he will not fail to make use of every opportunity offered him.

With some of the illustrations in this book the reader may already have made acquaintance. Beneath each borrowed illustration the source from which it has been taken, is recognised in the usual way. The author takes this opportunity of making grateful acknowledgement of his indebtedness to them.

80, RUSSA ROAD NORTH, CALCUTTA.

December, 3rd, 1913.

To
THE MEMORY
OF
MY FATHER
DR. JOGENDRANĀTH MUKHOPĀDHYĀYA
WHO FIRST DIRECTED
MY THOUGHTS TO THE STUDY OF MEDICINE AND
AIDED ME IN ITS PURSUIT
BY MUCH PRACTICAL INSTRUCTION
AND
WHOSE LONG PROFESSIONAL CAREER, SO USEFULLY
AND SO UNSELFISHLY SPENT
FOR THE POOR
HAS SERVED ALIKE AS A MODEL FOR IMITATION AND
AS A STIMULUS TO CONTINUED EXERTION,
I MOST GRATEFULLY, MOST AFFECTIONATELY,
DEDICATE THESE PAGES
AS A SMALL TRIBUTE.

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(In preparation.)

**THE STAR MEDICAL HALL,
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CALCUTTA.**

Tropical Abscess of the Liver.

CHAPTER I.

Importance of the subject. Materials. Anatomy of the liver. Classification of hepatic abscess. Synonyma. Definition. History of liver abscess. Geographical distribution of liver abscess and amebic dysentery.

The disease is of frequent occurrence in the tropics.

Importance of the subject. In India, it is so common as surely to come under treatment of every physician of ordinary experience. It is generally supposed that the "tropical liver is essentially a disease of the native of a temperate or cold climate residing in the tropics. It is a part of the white man's burden."¹ The truth, however, is that the natives of India are not exempt, though they suffer less than Europeans. It is common amongst those who indulge themselves in western luxuries. The etiology of the disease is still an open question; the relation of liver abscess to dysentery is not yet thoroughly known. The treatment also is far from being satisfactory; the disease often runs a fatal termination even under

¹ Albutt's system of medicine. Vol. II. Part II. P. 572.

skilful hands. A study of its etiology and pathogenesis may suggest indications for its prophylaxis and treatment. These are the reasons for selecting the subject as the theme of this thesis.

In preparing this article the writer has put together all Materials. the cases seen by him within the last twelve years, both in hospitals and private practice, as a student in the Medical College Hospital, Calcutta, as resident surgeon, Darbhanga Sadar Hospital, as a private practitioner, but who has regularly attended the Sambhunath Pandit Hospital, Calcutta, through the kindness of Lt.-Col. E. H. Brown, M.D., I.M.S., the surgeon-superintendent, for the last six years and as surgeon to the Albert Victor Hospital connected with the Calcutta Medical School and College of Physicians and Surgeons (Bengal), for four years 1902-06,—100 cases in all. The patients were all natives of India, the majority being Bengalis. All the cases accessible in the medical literature on the subject, have been gone over and noted. All the statements of this article are based upon an analysis of these cases, together with the consideration of the available literature on the subject. I have given a full description of the disease—my observations often corroborating the observations of others, but on a few points, where I differed, the divergences have been pointed out.

To understand the subject properly it would not be
out of place here to describe the anatomy
of the organ shortly.

Anatomy of the
liver.

The liver is situated in the right hypochondriac and epigastric regions, except in children where it extends across the epigastrium into the left hypochondriac region. Its weight varies from three to four pounds. If we calculate the quantity of blood which is lost when the gland is removed from the body, its average weight is four and one-half pounds—the physiological weight of Sappey. Its transverse diameter is from ten to twelve inches long, and the antero-posterior six to seven inches. The back of the right lobe, which is the thickest part is three inches thick. The upper surface is convex; it is covered by peritoneum and has the diaphragm above it. It is divided into two unequal parts by a fold of peritoneum called the suspensory ligament. The under or visceral surface is concave and is in relation with the stomach, the duodenum, the hepatic flexure of colon, the right kidney and the suprarenal capsules. It is also covered with peritoneum except where the gall-bladder is attached at the portal fissure, which gives attachment to the lesser omentum. The posterior surface is broad over the right lobe but narrow on the left. It is in contact with the diaphragm and the posterior wall of the abdomen. The posterior surface of the right lobe is rough and uncovered

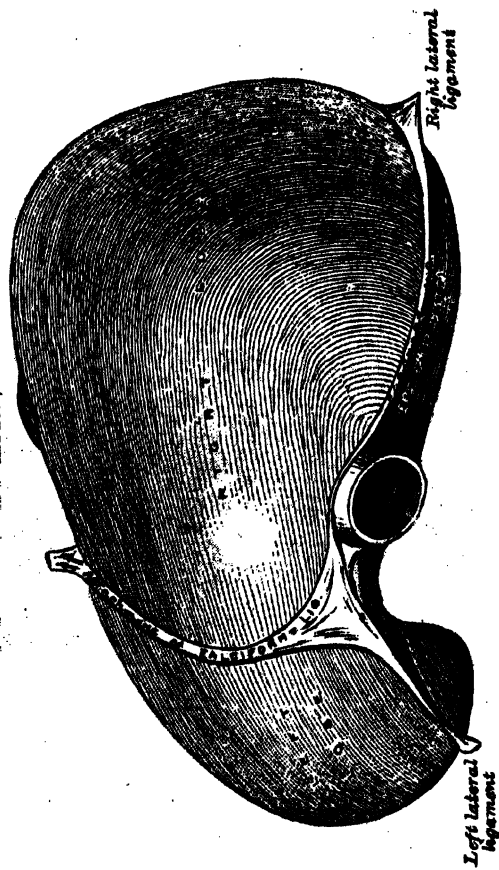
by peritoneum. The centre is notched for the vertebral column, and a little to its right is another indentation for the inferior vena cava. To its right again is the depression for the right suprarenal capsule; and to its left is a groove indicating the position of the œsophageal orifice of the stomach.

The anterior border is thin and sharp, and is marked opposite the attachment of the longitudinal ligament by a deep notch, and opposite the cartilage of the ninth rib by a second notch for the fundus of the gall-bladder.

The ligaments are five in number; four being formed of peritoneum. The longitudinal (suspensory, falciform or broad) ligament is attached above to the diaphragm, extending on to the sheath of the rectus muscle as low down as the umbilicus, and below from the notch in front to the posterior edge of the liver. The posterior edge encloses the round ligament. The lateral ligaments, right and left, extend from the sides of the diaphragm to the posterior border of the liver. The coronary ligament is continuous with the lateral ligaments and attaches the posterior margin of the liver to the diaphragm. The round ligament (obliterated umbilical vein) is contained within the posterior edge of the suspensory ligament.

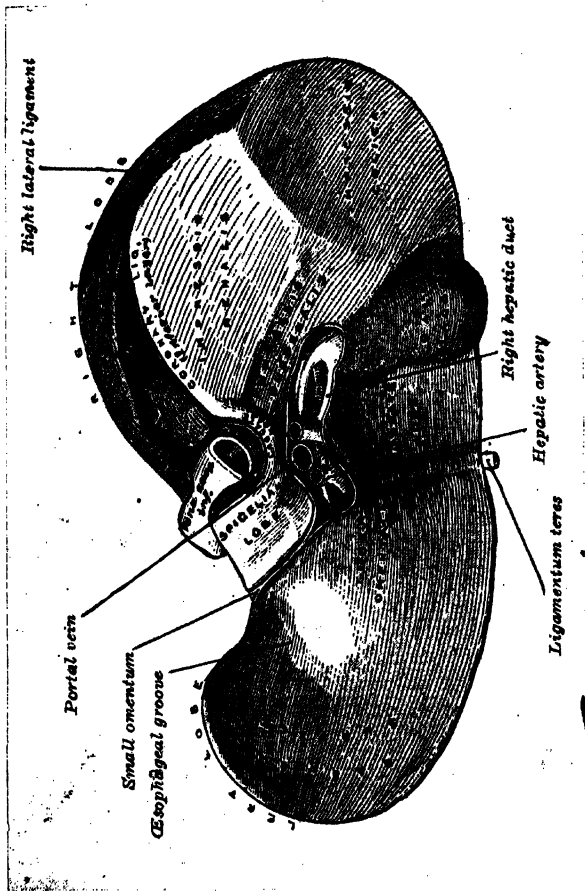
There are four fissures. The longitudinal fissure is occupied by the round ligament, and divides the

I. Superior Anterior Surface of the Liver.



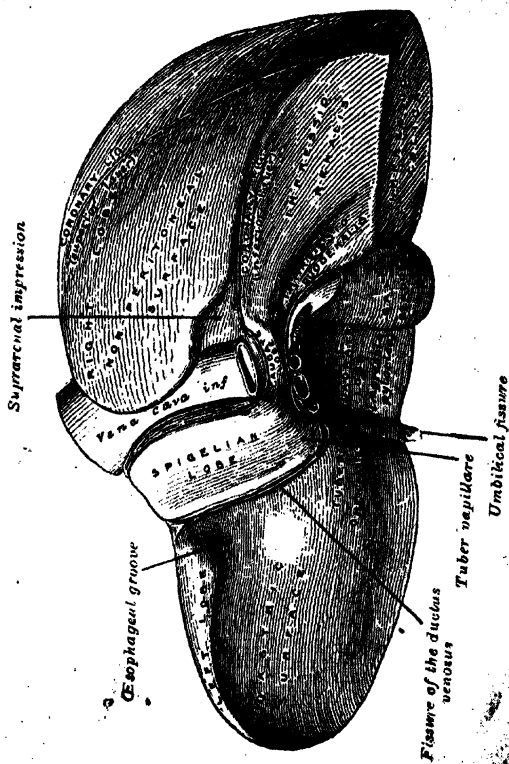
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2. Inferior Surface of the Liver.



To face page 5.

2 (a) Inferior Surface of the Liver.



To face page 5.

organ into the right and left lobes; it is divided into two parts by its union with the transverse fissure. The anterior part or umbilical fissure is between the left lobe and the lobus quadratus and contains the remnants of the umbilical vein. The posterior part or the fissure of the ductus venosus is between the left lobe and the lobus Spiegelii, and contains the remnants of the ductus venosus. The transverse or portal fissure is between the lobus quadratus and the lobus Spiegelii and lodges the hepatic duct, artery and portal vein. The fissure for the vena cava is at the posterior margin of the liver between the right lobe and the lobus Spiegelii, and is separated from the transverse fissure by the lobus caudatus. The hepatic veins enter the vena cava at the bottom of this fissure.

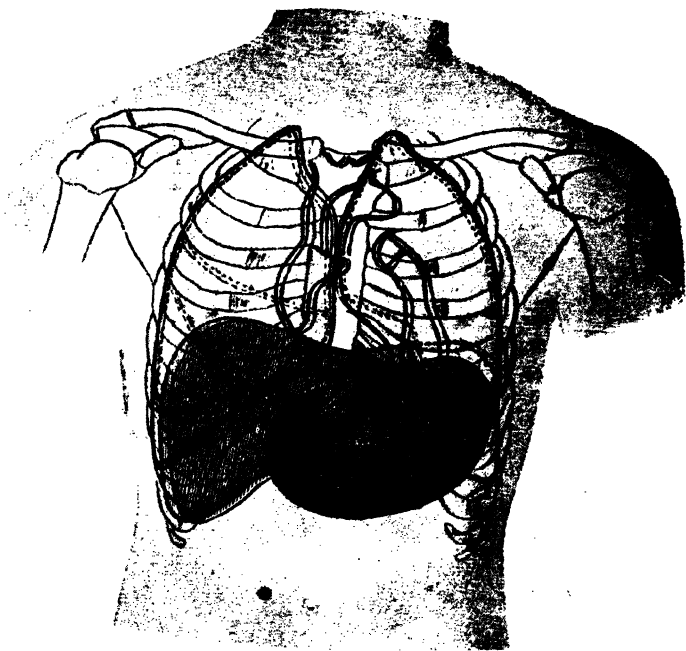
The right and left lobes are separated from each other
 Lobes. by the umbilical fissure on the under surface and posteriorly by the fissure of the ductus venosus. The right is the larger and contains the following lobes:—
 The lobus quadratus: bounded by the umbilical and transverse fissures and the fossa of the gall-bladder. The lobus Spiegelii: is the projection between fissures for the vena cava and ductus venosus and is behind the transverse fissure. The lobus caudatus: connects the lobus Spiegelii with the right lobe and is behind the transverse fissure.

The fossa for the gall-bladder lies on the under surface of the right lobe, parallel to the umbilical fissure, and separates the quadrate lobe from the main mass of the right lobe.

The vessels of the liver are five in number:—the hepatic artery, the portal vein, the hepatic duct, the hepatic vein and the lymphatics. They are arranged from right to left as follows:—the hepatic duct, the portal vein and the hepatic artery. They are enveloped in the capsule of Glisson and run through the portal canal into the organ. The hepatic veins have no cellular investment, so their parieties are adherent to the walls of the canal through which they run.

The nerves are derived from the hepatic plexus of the sympathetic, from the pneumogastric nerves, and from the right phrenic.

Surface form:—It lies under the lower ribs and their cartilages, but it touches the abdominal wall in the subcostal angle. The upper limit of the right lobe of the liver corresponds to a line drawn from the articulation of the fifth right costal cartilage to the sternum horizontally outwards to a little below the nipple, and then inclined downwards to reach the seventh rib at the side of the chest. The upper limit of the left lobe corresponds to the continuation of this line to the left with an inclination

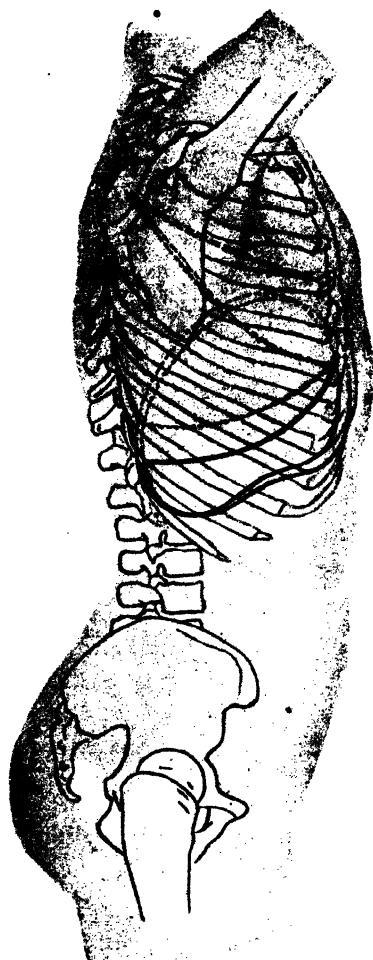


3. Situation of the Viscera (Anterior aspect)

Outlines of heart and vessels---broad red lines. Margins of lungs and individual lobes---dotted green lines. Limits of pleural sacs---solid green lines. Liver---red shading. Stomach green shading.

(In part after His-Spatteholz and Luschka.) Fevre.

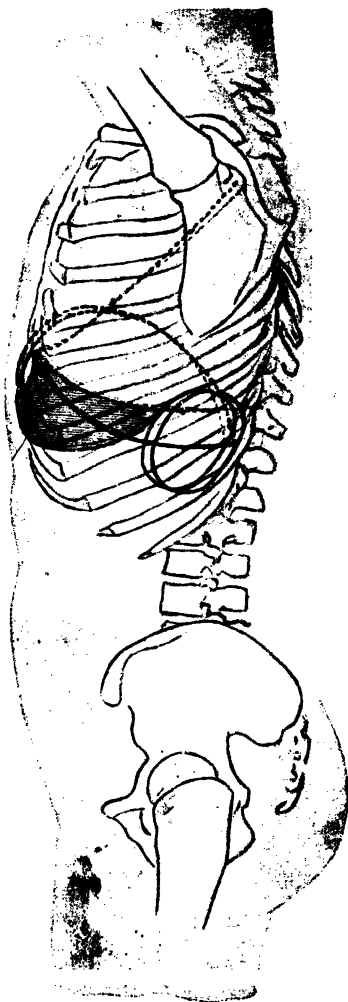
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5. Situation of the Viscera (Right Lateral Aspect)

Margins of lungs and of individual lobes---dotted green lines. Limits of pleural sacs---solid green lines. Liver and spleen---solid red lines. Diaphragm---dotted red lines. Stomach (portion not covered by lung)---green shading.

(In part after Luschka.)



6. Situation of the Viscera (Left lateral aspect)

Margins of lungs and of individual lobes---dotted green lines. Limits of pleural sacs---solid green lines. Liver and spleen---solid red lines. Diaphragm---dotted red lines. Stomach (portion not covered by lung)---green shading.

(In part after Luschka.)

downwards as it crosses the gladiolus, to a point about two inches to the left of the sternum on a level with the sixth left costal cartilage. The lower limit corresponds to a line drawn half an inch below the lower border of the thorax on the right side as far as the ninth right costal cartilage, and thence obliquely upwards across the subcostal angle to the eighth left costal cartilage. A slight curved line with its convexity to the left from this point to the termination of the line of the upper limit will denote the left margin of the liver. (Gray).

Roughly speaking we may define the extent of the liver thus: its lower border corresponds approximately with a line drawn from a point immediately below the lower extremity of the right costal margin to a point one inch below the left nipple. Connect these points with a point one and a half inches below the right nipple, by lines slightly concave towards the liver.

In Bengalis, the liver is lighter than that of Europeans, being 45 oz. on the average. Its transverse diameter is ten inches long and the antero-posterior six inches. The thickest part of the right lobe is two and one-half inches thick.

The relations of the liver to other organs and to the bony thorax will be apparent from the diagrams. "Above, the diaphragm, and at

Relations of the liver.

the pit of the stomach, the abdominal wall. *Below*, from left to right, the lesser curvature and part of the anterior wall of the stomach, the duodenum, the hepatic flexure, the right kidney (placed behind the colon). *Anteriorly*, the abdominal wall. *Posteriorly*, the diaphragm (separating the liver from the right lung and the thoracic aorta), the suprarenal capsule, and the inferior vena cava.”¹

Classification of Hepatic Abscess :

A. Etiological or Pathological :—

The liver is the seat of different kinds of abscesses, which vary clinically and pathologically.

1. Tropical Abscess. Hepatitis or Hepatitis Suppurative or Solitary Abscess :—Tropical Abscesses are not however always solitary although they are frequently so ; nor is any solitary large abscess of the liver (the so-called *Primary* abscess of the liver) confined exclusively to tropical countries. They are very common no doubt in hot climates, but they do occur, though rarely, in England² and other regions outside the tropics ; but in the temperate climate, abscess of the liver is invariably *secondary*. Again multiple

¹ A Text-Book of Naked-Eye Anatomy (Text to Masse's plates), by J. Cantlie. P. 405.

² Fagge's Text-book of Medicine edited by Pye-Smith Vol. II, P. 525, 4th Ed.

abscesses may unite and eventually form a large abscess. So the term tropical abscess probably is a misnomer.

2. Pyæmic, or Embolic, or Metastatic Abscess—also common, especially in Europe. These are caused by infective matter carried from other foci of suppuration into the liver. They are, as a rule, multiple and small.

In exceptional cases, a single hepatic abscess follows infection elsewhere in the body. Thus it has followed cutaneous abscesses, whitlow, parotitis, bronchiectasis (Muir¹), parametritis and pyosalpinx (Roughton²), prostatic abscess (Lancranux³), scarlet fever⁴ and influenza (Cimbali⁵).

3. Biliary Abscess or Cholangitis:—These are abscesses connected with diseases of the bile duct. Chronic diseases of the duct of the gall-bladder may lead to inflammation of the liver.

Abercrombie⁶, Bright⁷, Louis⁸ and Budd⁹ describe cases where the abscess of the liver co-existed with gall-

¹ Edin. Hosp. reports II. P. 100.

² St. Barthol. Hos. Rep. XXI. P. 176.

³ Traites's des maladies du foie et du Pancreas. 1899.

⁴ Guy's Hosp. museum no 1294.

⁵ Lo Sperimentale 1890.

⁶ Observations No. CXXVIII and CXXIX.

⁷ Guy's Hosp. Reports, Vol. I., P. 630.

⁸ Observations No. V.

⁹ Diseases of the liver, P. 92.

stones and irritation of the bile ducts. Intestinal worms sometimes penetrate into the biliary passages and produce abscesses. Kirkland¹ observed an intestinal worm escape with the pus from an hepatic abscess. Abscesses produced by intestinal worms are mentioned by Lobstein,² Lebert,³ Tonnelé⁴ and Forget.⁵ For details of these cases the reader is referred to Frerich's Diseases of the Liver.⁶

4. Suppurating Hydatids:—Not common here.

5. Traumatic:—Abscess of the liver may be caused by injury. This is very rare. Of 62 cases collected by Budd, there was history of violence in 2; Morehead out of 318 observations could trace this cause in 4.

Traumatism may give rise to hepatic abscess in two ways: (1) directly: when a penetrating wound by a dagger, or bullet, introduces micro-organisms into the liver. Rarely a fish bone, or a pin may penetrate the alimentary canal, enter into the liver, and cause suppuration there. (2) Indirectly: by so reducing the resistance of the liver and thus making a fit soil for pus-forming micro-organisms, intro-

¹ An Enquiry into the present state of Medical Surgery, 1786.

² Journal Complement. Tom. XXXIV, P. 271.

³ Traité d'Anatom. Pathol. Tom. I., P. 412.

⁴ Journ. Hebdom., 1829, Tom. IV.

⁵ Union Médic., 29 Mai, 1856.

⁶ Vol. II, P. 118. Syd. Soc. Trans.

duced by the hepatic artery or the alimentary canal. Turner¹ referred only to 12 cases. It is not common. Again a latent abscess may increase and by leakage set up a perihepatic or subphrenic abscess.

6. Abscesses from foreign bodies :—Also rare. Some foreign body is introduced into the liver, carrying with it streptococci and other organisms and so large abscess results.

7. Suppurating tubercular and cancerous growths :—They are also rare.

This etiological or pathological basis of classification has not met with universal approval, “for the tropical abscess may be pyæmic in origin, and with the tropical are associated abscesses occurring with, or following upon, dysentery, the connection of which with a pyæmic infection of the liver is a matter largely of opinion” (Cantlie).² So he brought forward another basis of classification :—

B. The Anatomical :—

a. Suprahepatic abscess :—collection of pus between the layers of the broad (coronary) ligament of the liver.

b. Intrahepatic abscess : abscess within the liver substance.

(1) by Inflammatory changes *within* the liver.

¹ Trans. Path Soc. vol. XXXIII, P. 177. 1832.

² Encyclopædia Medica. vol. 7, P. 18-19.

- (a) Hepatitis ending in suppuration or tropical abscess of the liver.
 - (b) Traumatic.
 - (c) Suppurated hydatid and other cysts.
 - (d) Parasitic.
 - (e) Inflammatory hepatic lesions caused by gall-stones, pyelephlebitis, and other diseases.
- (2) by causes *beyond* the liver, but *within the gathering area of the portal vein*. Pyæmic :—
- (a) Dysenteric.
 - (b) Appendicitis.
 - (c) Diseases of, and surgical operations upon, parts drained by the portal vein.
- (3) from a general pyæmic state, the infection being conveyed by the hepatic artery : multiple pyæmic abscess of the liver. Dysenteric abscess.

c. Subhepatic abscess : collection of pus between the under surface of the liver and the peritoneum.

The hepatic abscesses have, from an anatomico-pathological point of view, been divided into four varieties ; so the third method of classification of the liver abscess is :

C. Anatomico-pathological :—

- 1. Metastatic or pyæmic abscesses.
- 2. Single phlegmonous abscesses (Kelsch and Kiener) :

It is the variety ordinarily known as tropical abscess of the liver related to dysentery.

3. Multiple fibrous abscesses (Kelsch and Kiener) : They are small and multiple. The development of the fibrous structure due to an inflammatory proliferation of the hepatic connective tissue may be strengthened by a deposit of lime salts and may thus arrest the suppurative process, without causing important morbid phenomena.

4. Alveolar abscess (Chauffard) : It is deep-seated and develops towards the surface in the form of an infarct. These abscesses are the result of partial suppurative angiocholitis, and are caused by the migration through the biliary passages of a septic germ which may travel upwards and induce the lesions of areolar abscess around and above whatever point it becomes fixed upon.¹

Hepatic Abscess. Tropical Abscess. Hepatitis or Synonymn. Hepatitis Suppurative. Solitary Abscess. Intra-Hepatic Tropical Abscess. Fr. Abscess du Foie. Gr. Leberabscess.

By "Tropical abscess of the liver" we mean the Definition. single or multiple large abscesses whether dysenteric or non-dysenteric (idiopathic) which are commonly met with in the warm climate.

¹ Twentieth Century Practice of Medicine, vol. ix, P. 557.

History of
the liver
abscess.

Abscess of the liver is mentioned by Caraka and Suśruta who flourished about 1000 B.C. Hippocrates directs that abscesses of the liver are to be opened by the cautery¹. He states that abscesses of the liver are least dangerous when they open externally ; more so when they open internally ; and most of all when they open both internally and externally². Galen approves of venesection and prescribes detergent medicines ; purgatives are indicated when the concave side is the seat of the disease and diuretics when the convex side is the seat.³

Aretacus accounts for the pain which is felt at the top of the shoulder or clavicle in this way : the liver being enlarged and becoming heavier than natural, drags down the diaphragm to which it is attached, and thus stretches also the pleura from its upper adhesions, whereby pain in the part is produced.⁴ When pus is formed, it may be discharged by urine, by the bowels or it may point out outwardly. In this last case, he advises us to open it with a red-hot instrument. Celsus recommends bleeding, purging, diuretics and when a vomica forms, it is to be

¹ Coac. 457.

² Prognost 7.

³ Galen's *Methodus Medendi*, xiii, Sec. Loc. vii.

⁴ *Morb. Acut.* ii. 7, *Morb. Chron.* i. 13.

opened and burnt¹. Caelius Aurelianus² the methodist, justly condemns as too bold and dangerous a measure, the proposal of Erasistratus to lay bare the liver, and apply remedies direct to the part affected. Both Avicenna³ and Serapion⁴ make mention of taraxacum as a remedy. Haly Abbas⁵ recommends diuretics; and when the abscess bursts into the cavity of the peritoneum he advises us to open it. When it makes its way to the stomach, or the bowels, he prefers purgatives. Rhazes⁶ also explains the pain at the shoulder in the same way as Aretaeus. For the opinions of the other ancient authors, see the references mentioned below⁷.

The liver abscess is essentially a disease of
 Geographical the tropics, though no climate seems to be
 distribution. exempt.

Europe. It is rare in the temperate and cold climates. In England, it is rare. But Fagge⁸ doubts the correct-

¹ Celsus, iv. 8.

² Caelius Aurelianus, Pass. Tard. iii. 4.

³ Avicenna, iii. 14.1.

⁴ Serapion, iv.

⁵ Haly Abbas, Theor. ix. 30; Pract. vii. 31.

⁶ Rhazes, Divis. 62; ad Mansor ix. 67; Conties. xvi.

⁷ Alexander vii. 19. Aetius X. 1. Oribasius Loc. Affec. iv. 96. Nonnus (182), Octavius Horatianus ii. 13. Marcellus De. Med. 22, Scribonius Largus 30, Avenzoar l. 13, Alsharavius Pract. xviii.

⁸ Fagge's Medicine. Vol. II. P. 358. 3rd Ed.

ness of this statement. Of the 15 cases which proved fatal in Guy's Hospital he found that "only five of these cases occurred in persons who had come from China, or India, or the West Coast of Africa ; but in 10 there was no such history, and most of the patients, it was positively stated, had never been out of England." Cases occurred in Ireland (1818-26), Dublin (1818) and the Lancaster Asylum before. France, Germany, Northern and Central Europe are now quite free, though formerly cases were far from being rare there. It is present in Roumania, Italy and Dalmatia. In Gibraltar the mortality is 0.11 per 1000, in Malta 0.43 per 1000. In Cyprus it is rare though dysentery is common. It is more frequent in Greece, Turkey, Southern Italy and Southern Spain.

Asia. It is very common in India : in Bengal (death rate 1.35 per 1000), Orissa, Gangetic plain, Chota Nagpur, North-Western Frontier, Indus Valley, North-West Rajputana, South-East Rajputana, Central India, Gujrat, Deccan, Southern India, West Coast and the hill stations. It is most frequent in the Madras Presidency (death-rate 6.3 per cent.). It is very common in Burma (2.30 per 1000) and Malacca (Stewarts). It is rare in Ceylon where bacillary dysentery is the type, but Marshall reports its common occurrence there (5.5 per cent. of the total strength). It is less common in Malaya Peninsula. Penang and



Red lines---Dysentery (amœbic and bacillary), Green lines---Tropical Liver Abscess.
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Straits Settlements are free. It is common in **Cochin China** and **Tonkin** (death rate of the French soldiers 2 to 3 per cent.). But the French authorities state it to be rare there (Foiret, Richaud, Saux and others.) It is absent in **South China**. In **Hong Kong** and the **Chinese Ports** (Wilson) it is rarely met with, though dysentery is common. So in the southern-most parts of **Japan** (Kommamoto in Sikokf, and Kiusiu and Nagasaki) where bacterial dysentery is the prevailing form. It is rare in the northern parts of the empire. It is more frequent in **Asia Minor**.

In the *East Indies*, the coasts of **Java**, **Sumatra** and **Borneo** (Heyman) and the island of **Phillipines** (Sollaud) are the chief seats of the disease.^m It is more rare in **Banka**, **Celebes**, the **Moluccas**, the **Riouw-Lingga Archipelago**, and the **Andamans** (Douglas). [It is also rare in *Polynesia*, the **Hawaiian Islands** (Gulick) and **Tahiti** (Dutrroulau). It was rare in **New Caledonia** before, but there it has become common lately (Rochas).

It is very prevalent among **Europeans** in the tropical parts of **Arabia** and **Persia**, especially along the the coasts of the **Red Sea** and **Persian Gulf** (Pruner).

In **Madras**, it is oftenest prevalent on the **Coromandel** and **Eastern Ghats**, **Bangalore** (11·10 per cent.) and **Secun-**

derabad (14·54 per cent.). It is less common in Cochin (Day).

In Bombay, Morehead estimates the number of cases to be 7·4 per cent. of the total strength of the British troops; it is on the slopes of the Ghats that the malady is most common (Hunter).

It is less common in Bengal, the annual number of cases being 5 to 6 per cent. of the total strength (Macpherson). It is especially common in some parts of N. W. Provinces (McGregor).

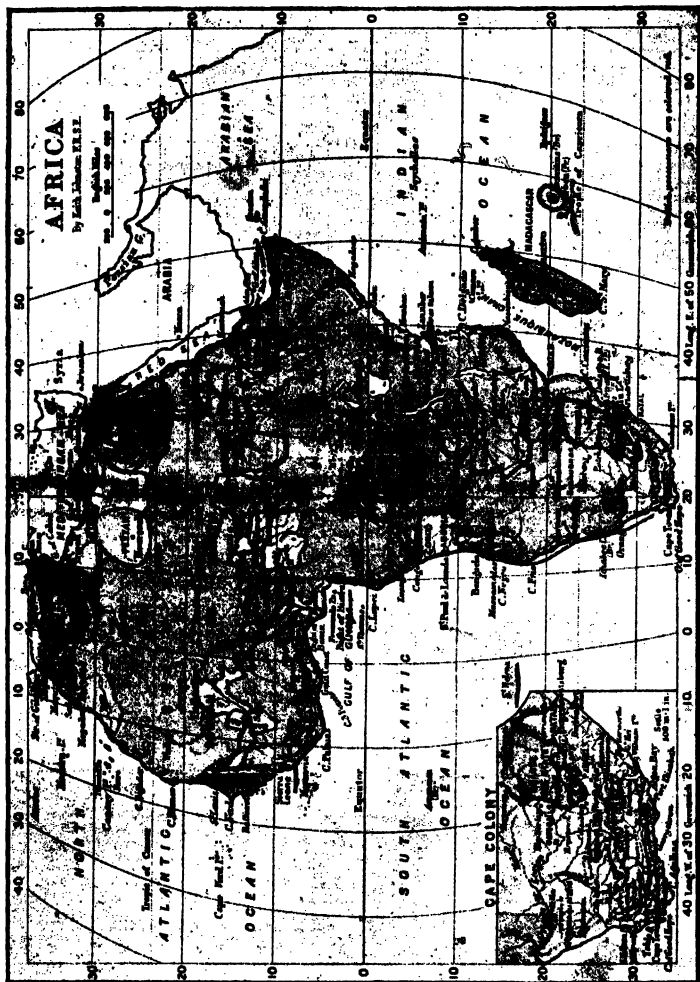
The following are the figures of the cases of hepatitis among the British troops :—

Fayrer :—

Presidency.	Years observed	Death Per 1000.
Bengal.	1850-- 1879	2·04
Bombay.	„	1·71
Madras	„	3·31

Cheevers :—

Presidency.	Years observed.	Per 1000 of all admissions.
Bengal	43	2·9
Bombay	51	3·8
Madras	12	6·3



Morehead :—

Race.	Years.	No. of cases of hepatitis admitted.	Mortality.
European Civil Hospitals, Bombay.	6	3·7 per cent of total admission.	7·8 per cent.
Native „ „		1·5 „ „	3 per cent.

Africa. It is common in Nubia (Pruner), Egypt (1·0 per 1000) (Sachs), Algeria (Cambay) especially in Oran, Philippeville, Bougie, Senegal (12·4 to 9·6 per 1000), Madagascar (Borchgrevink) and Mozambique (Roquette). Rare in Sierra Leone and Lagos (Macfarlane) though dysentery is common. It is common in the Congo Free State as result of dysentery (Dryepondt) and in St. Helena (death rate from hepatitis 29 per 1000) (Lombard). Zanzibar is quite free (Drago) and it is rare in Transvaal. It is common in Mauritius (Allan), and Réunion (Pellissier). It is very common in Senegambia (Thevenot), the Bight of Benin and Bight of Biafra (Boyle), the Slave Coast (Féris), the Gold Coast (Clarke) and Fernando Po (Quetan).

America. It is frequent in New Orleans (Sullivan); rare in the West India Islands, St. Thomas (Barclay), Barbadoes (Jackson), Martinique (Rufz), Guadeloupe (Olméte), Jamiaca (Ross) Trinidad and the Leeward Islands, though dysentery is quite common in these places.

It is more common in Domingo (Desportes) and Cuba (Sullivan) as a complication of dysentery but not exclusively so. It is rare in British Guiana, Demerara (Blair), Surinam (v. Leent), and Cayenne. It is common in Brazil (Sijaud) but Dundas reports it to be rare there. It is endemic and extremely fatal along the northern coast of Chili though enjoying a temperate climate (Murillo) and coast of Peru (Tschudi). It is also common in Venezuela (v. Archen).

Central America: It is of frequent occurrence in Panama, Costa Rica, Guatemala and Salvador. The West Coast of Mexico is more subject to it than the East Coast, but generally it is rare there.

United States: It is rare there. Acute hepatitis is uncommon even in the Southern States (Nott and Little). It is rare in northern states but more frequent in the south (Osler).

Australia: In Australia hepatic abscess is met with, but far less frequently than in India (Waring).

The northern coast of the Gulf of Mexico forms the limit of hepatic abscess in the Western Hemisphere, and the Mediterranean form its limit in the Old World (Hirsch). Thus we see that the hepatic abscess is not met with uniformly in different parts of the tropics: while it is frequent in India, Senegal, Ceylon, Mauritius, Algiers, Egypt, Java



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Sumatra, Mexico, Peru, Chili, it is much less common in the West India islands, China, Brazil, and Guiana. Both Lombard and Hirsch maintain that "hepatic abscess does not occur frequently, in the corresponding parallels of latitude in the United States, which is true of the Atlantic border, but is not correct of the interior continent, the valley of Mississippi, and its tributaries".¹

Table of the relative frequency of hepatites, congestion, and abscess of the liver in European soldiers in foreign stations (Nash.)²

STATION.	Years.	Strength.	Disease.	Admitting.	Died.	Invalided.
Bengal	1888-90 (3 years.)	125,371	Hepatites, Congestion and liver abscess.	2831	169	178
Madras	1888-91 (4 years.)	54,440	"	1018	97	102
Bombay	1888-90 (3 years.)	37,589	"	517	36	65
Ceylon	1888-91 (4 years.)	4,558	"	66	4	3
West Indies (White)	"	4,340	"	14	1	0
Bermuda	"	5,813	"	13	0	1
Canada	"	5,556	"	12	0	1
Gibraltar	"	18,938	"	29	2	2
Malta	"	27,621	"	182	12	6
China	"	6,441	"	96	2	4
Straits Settlements	1889-91 (3 years.)	3,584	"	24	0	3
Mauritius	1889-91 (4 years.)	2,025	"	22	3	1
South Africa	"	13,260	"	60	3	12

¹ Pepper's System of Medicine, Vol. II. p. 1003.

² This table is condensed from the information supplied by Brigade-Surgeon Lieutenant-Colonel Nash, Army Medical Staff as quoted in Davidson's Diseases of Warm Climates, P. 647-8.

As dysentery (amœbic) is commonly adduced as the sole cause of tropical abscess of the liver, let us see how far the geographical distribution of amœbic dysentery. distribution of dysentery corresponds to that of liver abscess.

Europe. Amœbic dysentery occurs sporadically in St. Petersburg, Konigsburg, Kiel, Hamburg, Berlin and other towns in the north of Europe. It is rare in England but more frequent in the Central and Southern regions of Europe—Italy and Greece. Jäger reports a moderate epidemic in the German army in East Prussia 1901.

Asia. It is very common in India ; but seldom met with in the upper provinces (Duncan). More common in Calcutta though the bacterial variety is the prevailing type. It occurs in Siam, Indo-China and parts of China. It is very prevalent in the Philippine Island (Strong and Musgrave) and forms 67 per cent. of the total cases of dysentery. In Manila it forms 80 per cent. of the cases and more than 30 per cent. of the white men suffer there. It is more common along the coast line of India than the interior but contrary is the case in Ceylon (Fernando) where it is rare (Castellani).

Africa. Egypt is the home of the disease. It is frequent in Algeria and Senegal. No part of Africa is



exempt, but the bacillary variety predominates on the West Coast. In South Africa, it is rare (Beveridge). It is endemic in Mauritius.

America. Isolated cases occur in Michigan (Dock) and Philadelphia (Musser). More numerous in the middle and southern states. It is the commonest variety in the United States (Osler). It is not very common in the British, Dutch and French Guiana; more commonly met with in Cuba, Martinique, Rio de Janeiro, and other parts of Brazil and North of Chili. In North America, it is common in districts approaching the sea level—as the shores of Chesapeake bay, Gulf of Mexico and Mississippi valley.

Thus we see that liver abscess is most common in countries where amœbic dysentery prevails. Manson¹ says: "The geographical ranges of liver abscess and dysentery concur, therefore, only in warm climates, and in them only to the extent that liver abscess is rare or unknown in places where dysentery is rare or absent." But its absence does not necessarily prove the absence of dysentery, and it is also present in countries where amœbic dysentery does not occur. In the West Indies, although dysentery is common, liver abscess is relatively rare.

A glance at the map will at once show that the areas marked for their prevalence do not wholly coincide.

¹ Gibson's Text-Book of Medicine, Vol. I, P. 266.

CHAPTER II.

Etiology of liver abscess. Hepatitis. Altitude. Tropical climate. Meteorological conditions:—1. High mean temperature. 2. Vicissitudes of temperature. 3. Seasonal prevalence. Liver abscess and dysentery in Calcutta hospitals. Relation of liver abscess to dysentery.

Acute and chronic hepatitis is more commonly met with in tropical regions than in temperate zones. And hepatitis often plays a significant part in the etiology of liver abscess. It is caused by the injection of rich food and stimulating drinks with deficiency of exercise. But as Europeans who habitually over-eat and drink in temperate climates, and who also suffer there from hepatitis, do not equally suffer from liver abscess, there must be some local causes in determining the ailment in the tropics.

The influence of altitude as moderating the temperature, and so the prevalence of liver abscess, can not be doubted. In Algeria, it is rare at an elevation of 3000 metres (Rouis); it is absent at high

elevations in Mexico and Peru (Jourdanet and Tschudi).

But it is often more frequent at moderate elevations than at the sea level; and so at Secunderabad, 1700 ft. above the sea level, fatal dysentery, with liver abscess, was very common untill the introduction of sanitary improvements in recent times. The following table shows clearly the influence of altitude (Davidson):—

			Elevation in feet.		
			Below	100 ...	2·8.
			100 to	500 ...	2·4.
1895 ...	} European army in India	Admission per 1000 for liver abscess.	500	1500 ...	1·7.
1896 ...			1500	3500 ...	2·1.
1897 ...			3500	5000 ...	1·9.
			5000	8000 ...	1·2.
			8000	13000 ...	0·0.

Even in elevated localities it may be common, as we know of its more frequent occurrence on the slopes of the Ghats in Madras (Balfour) and Bombay (Hunter) Presidencies than on the plains of Bengal. Great influence is attributed to the heat of the climate in

causing inflammation and abscess of the liver. A hot climate, no doubt, deranges the functions of the liver and causes increased secretion of irritating bile. But heat of itself does not appear to be the principal cause of the disease (McGregor). Its frequency within equatorial countries varies under the same sort of climate; it is more prevalent in Mauritius, Algiers and Chili with a comparatively mild climate, than in the highly tropical countries, as

Jamaica, Guiana and the south of China. It is more frequent in Madras than in the highly tropical Bengal. It has also been found that the disease touches its maximum, not in the hot season, but in the colder rainy season.

The heat is certainly not the only factor in its *causal nexus*, as sailors employed in the trade of India do not suffer; nor do those who by their trade are exposed to high temperature, as glass-blowers etc. (though cases are on record where such men developed liver abscess). It is on the continuity of the cause, chiefly acting in conjunction with other excesses, that we must seek for a solution of the problem. The liver has more work to do, and this additional work is then remotely the cause of liver abscess in a man, whose liver has been rendered inadequate by alcoholism and attacks of dysentery. Again heat depresses the vasomotor mechanism, thus exposing the man to be affected by a chill, to which Europeans are especially liable in India from the constant use of warm clothing and by fatigue induced by over-work. The Indians undertake hard work in the morning and evening and take rest at noon; and they always use light clothes;—habits which have been acquired instinctively. Consequently they do not suffer so much from heat like the inhabitants of cold climate residing in the tropics. As the natives do not suffer and as the European females and children are not

liable to the disease, though equally exposed to heat as the men, tropical climate can not be held solely responsible for the occurrence of liver abscess.

The influence of high mean temperature in determining abscess of the liver can be easily inferred from its geographical distribution. Rouis observed that in 1843, 1847, 1849 and 1853, the temperature was high in Algeria, and the cases of liver abscess were also more numerous. Graves relates a case of liver abscess in a glass-blower, a trade in which the workmen are subjected to great heat. The liver is excited by more secretory work falling on it in hot than in temperate climates, some of the effete materials which would in temperate regions pass away by the lungs, being removed from the system by an increased flow of bile. But the effect of high mean temperature is not uniform as will be apparent from the following table compiled by Mouat for the military stations in the Madras Presidency¹ :—

Table of admissions for hepatitis, with the mean temperature.

Station.	Mean temp. of the year.	Admission per 100 men.
Trichinopoly	84° F	8·7
Wallajabad	83° „	1·7

¹ Madras Quart. Journals. 1839.

Station.	Mean temp. of the year.	Admission per 100 men.
St. Thomas Mount ...	83° F ...	4·7
Masulipatam ...	83° „ ...	4·9
Madras ...	83° „ ...	7·4
Bellary ...	83° „ ...	8·2
Secunderabad ...	81° „ ...	14·5
Arni ...	81° „ ...	5·7
Arcot ...	81° „ ...	13·5
Cannanore ...	80° „ ...	8·7
Bulgam ...	76° „ ...	9·2
Bangalore ...	74° „ ...	11·1

Another effect of heat is to favour the growth of amœba, so intestinal amœbiasis becoming common, may set up abscess of the liver.

Tropical climate no doubt determines the incidence of hepatic abscess. But heat is only one factor of tropical climate and the other meteorological condition must be considered, *e. g.*, the cold at night.

From solar exposure, heat and consequent excessive perspiration, the skin is rendered irritable and weakened, and is therefore more susceptible to variations of temperature, which, both seasonal and daily, are rapid and great. Hence the surface of the body being

2. Vicissitudes
of temperature.

easily chilled, the internal organs, especially the liver, in its excited condition, suffer much in the same manner as in the "cold stages" of fever. We thus find the comparative rarity of liver abscess in Straits Settlements and British Guiana with a hot, moist but equable climate as compared with Senegal and Chili, where there are fluctuations in the high temperature. The equatorial belt is the area of equable temperature. There the diurnal and nocturnal fluctuations in temperature vary but a few degrees, and consequently chill is uncommon ; and we find liver abscesses to be as rare there as in the cold climate. But in India, the climate is marked by fluctuations in temperature, which exerts potent influence upon the prevalence of the disease. The Europeans after their return from the tropics sometimes develop abscess of the liver due to the inclemency of weather. Change of temperature, Maclean attributes as a cause not only of acute congestion of the liver, "but of fatal suppurative inflammation" Davidson mentions a case in Mauritius, where an intemperate Hindu developed liver abscess, by sleeping on the damp ground, during night¹. "At stations where the days are very hot," says Murray, "and the nights cold, or where the transitions of temperature are great, sudden and frequent, there will be more hepatitis in a corps than where the temperature is uniformly high."

¹ Albutt's System of Medicine, Vol II, Part II, P. 584.

In the previous history of patients suffering from liver abscess, we often note chill as the exciting cause. One of the causes why the European soldiers and sailors suffer so much, has been described by Johnson and Martin¹ to be that when "exhausted by exercise in the heat of the day and by profuse perspiration, he strips himself the moment his duty is over and throws himself down opposite a window or post, to inhale the refreshing sea breeze; his shirt in all probability, dripping with sweat." The thermometer at Bombay and Calcutta, in January is frequently as low as 55° in the night, and in April indicates up to 90°, or higher in the day; making an annual vicissitude of 35°. Sir James McGregor² in his report to the Medical Board at Bombay, for November 1800, observes that the mercury had an extraordinary wide range from 50° to 130° in the open air. And he shortly afterwards adds—"More cases of hepatitis appeared than in either of the two former months."

Twining remarks: "The most common exciting causes of hepatitis appear to be diurnal alternations of temperature, which occur in Bengal at the beginning of the cold season." In Senegambia, the disease is at its maximum in the season when the temperature during the day rises to 104°F, but

¹ The Influence of Tropical Climate on the European Constitution, Page 234.

² Edin. Med. and Surg. Jour. 1805, Page 271.

during the night falls to 68°F, on sometimes to 50°F in the morning. Many authorities—Larrey, Johnson, Morehead, Vauvray, Vidaillet, Murrillo, Boyle and others—regard this extreme diurnal range of temperature as a causal factor in the etiology of liver abscess; but there are surely other factors at work, to cause the varying prevalence of the disease in localities which to all appearances are equally exposed to similar conditions of temperature.

Liver abscess is very frequent after rainy seasons, and just at the beginning of the cold season when the range of temperature is greatest. [Rogers denies any seasonal prevalence in Calcutta, his cases being indifferently distributed throughout the year. But the other Indian authorities—Twining, Murray, Geddes, Annesley, Waring and Morehead—agree “that the maximum falls at the end of the rains and in the cold season (according to Morehead in February and March, and in November, December and January).” (Hirsch).

Waring... Madras... 243 fatal cases

101 or 41·6 per cent in cold season.

79 or 32·5 „ „ in rainy „

63 or 25·9 „ „ in hot „

Cochin China... 51 cases

48

in rainy „

Thevenot...Senegambia...51 cases

31	from October to March.
11	„ April to June.
9	„ July to September (hottest period.)

The majority of my cases occurred during the rainy season. Thevenot, at St. Louis in Senegal, dates 31 out of 51 cases from October to March, in the cold season.

The relation of liver abscess to dysentery will be apparent from the following table which

The number
of cases of li-
ver disease
and dysentery
treated in the
Calcutta hos-
pitals.

shows the statistics of admission of the two diseases for twenty years in the Calcutta hospitals. It will be seen that diseases of liver, not to speak of liver abscess, are very

disproportionate to the number of cases of dysentery treated in these hospitals. Again it is evident that the proportion of liver diseases to dysentery in the Campbell, Police and S. N. P. Hospitals, where Indians are treated is remarkably less than that of the Medical College, Howrah General and the Eden Hospitals where both Indians and Europeans are treated and the Presidency General and Ezra Hospitals where the patients are exclusively Europeans and Jews. This shows that dysentery though very common amongst the natives, does not give rise to liver abscess amongst them in proportion to what happens in the European community.

Hospitals.	1883	1884	1885	1886	1887	1888	1889	1890	1891	1892	1893	1894	1895	1896	1897	1898	1899	1900	1901	1902	1903
I. Medical College	Dysentery.																				
	Liver disease.																				
	142																				
	109	148	138	139	137	172	170	128	871	611	806	889	818	765	753	562	794	833	721	783	685
II. Eden		141	135	117	103	117	173	172	491	596	554	736	561	745	695	395	590	526	468	414	395
															68	51	25	24	40	84	75
															38	25	7	7	17	9	20
III. Kara														40	17	6	12	8	6	7	9
														52	48	2	3	5	1	1	3
IV. Presidency General	108	91	162	111	116	84	104	73	94	45	54	96	106	99	60	125	121	93	118	97	102
	59	64	53	57	48	52	57	61	66	49	47	59	58	74	53	50	84	86	86	50	59
V. Campbell	496	556	831	733	834	693	751	622	647	820	1420	1700	1662	1945	3118	1242	1198	1695	1422	1306	1203
	15	16	18	21	17	68	77	40	75	197	290	262	241	202	364	285	296	380	260	341	371
	151	168	169	164	142	157	103	132	571	467	544	637	705	433	632	424	310	402	538	532	448
VI. Howrah General	16	18	20	23	28	29	14	11	84	123	146	106	145	135	173	124	134	164	132	174	146
	346	322	374	303	365	243	235	192	276	167	225	228	244	208	390	213	390	503	319	283	296
VII. Police	1	7	10	16	11	22	12	12	7	4	5	6	11	5	2	4	6	3	4	9	9
									263	135	197	170	180	301	680	316	558	890	600	666	648
VIII. S.N. P.									2	96	45	80	71	89	192	101	157	215	183	127	133

The relation of liver abscess to dysentery has been observed from the early times. Budd first propounded the theory that the liver abscess is always due to purulent absorption from the dysenteric bowel. Macnamara says: "I think there can be no doubt as to the fact of the very common association of liver abscess with dysentery,—the nature, however, of their connection is uncertain." Waring's table shows the proportion of cases of hepatic abscess occurring after or during the progress of various diseases :—

	No.	per cent.
Hepatitis, acute and chronic ...	131	or 43·666
Dysentery, acute and chronic ...	82	„ 27·333
Dysentery and hepatitis, or hepatic dysentery ...	14	„ 4·666
Fever or common continued fever ...	14	„ 4·666
Intermittent fever ...	5	„ 1·666
Remittent fever ...	3	„ 1·000
Diarrhœa or purging ...	6	} „ 2·333
Diarrhœa and intermittent fever ...	1	
Admitted with hepatic abscess evi- dently formed ...	6	„ 2·000
Catarrh and catarrhal fever ...	2	„
Delirium tremens ...	2	
Rheumatism ...	2	
Abdominal inflammation ...	2	

	No.	per cent.
Consumption	2	
Constipation and a swelling simulat-		
ing ventral hernia	1 „	6·333
Injury of the side from contusion		
or fall	3	
Ulcer on the leg followed by dysentery	1	
Disease of the brain	1	
Acute ophthalmia	1	
Disease of the knee-joint	1	
Pneumonia (?) following unusual exertion	1	
Doubtful	19 „	6·333
	300	100·000

Here we find that out of 300 cases of tropical liver abscess there was history of dysentery in 96 only. Even supposing that in many of these cases the symptoms of dysentery were latent, it needs explanation how only in 82 cases or 27·3 per cent. only was hepatitis preceeded by symptoms of dysentery.

We have the evidence of other observers also that dysentery does not commonly cause liver abscess as one of its complications. In Germany, Heubner¹ reports that hepatic

¹ On Dysentery. *Ziemssen's Cyclop. of Med. Amer. Ed. 1875. I. 546, 556.*

abscess sometimes occurs as a complication of dysentery, but they are always multiple and of embolic source; and it is remarked that "those abscess must not be confounded with tropical abscess." Cameron¹ says that

Observer.	Place.	Year.	No. of cases dysentery.	No. of cases liver abscess
Waring	Madras	1826-43	2758	68 or 2.5 percent
Finger	Prague	1846-48	231	0
Altschul	"	1880-84	279	1
Marston ²	Malta, Crimean war		Numerous	2
Burkardt	Franco-German war			rare
Baly ³	Nancy		80 fatal cases	0
Broussais	Millbank Penitentiary	1840-47	Many	0
Davidson	France	1805-06	17	0
Washbourn ⁴	Boer War	1899-02	Numerous	rare
Richards	Imperial Yeomanry	"	252	1
Buchanan ⁵	Hospital		466	1
Rokitansky	India prisons		681	1
Wilson ⁶	China		Dissected many cases	rare
Chakrabarti ⁷	Medical College, Calcutta.	1864-65	Common	"
Gemmel ⁸	Lancaster Asylum		280 (41 post mortem)	3
			80	2

¹ Med. Times and Gazette 1853. Oct. 365.

² Medical Times. September 1856.

³ Gulstonian Lecture on Dysentery 1847. Med. Gaz. Vol. IV. P. 885.

⁴ Washbourn and Richards, B. M. J. 1900, II. p. 668.

⁵ Buchanan W. J. Journ. Trop. Med. Vol. I. p. 173. 1899.

⁶ Dr. Wilson in the Records of Hospitalship "Minden."

⁷ Dr. S. J. Chakrabarti in Indian An. of Med. Sc. 1865 Page 150.

⁸ Gemmel: Idiopathic Ulcerative Colitis P. 24, 1898.

dysentery is rarely complicated with abscess of the liver in Ceylon.

Abercrombie¹ regards the association of liver abscess with dysentery as "accidental combinations, but independent of each other, though they may considerably modify the symptoms". Murchison, Macpherson and others concur with this view.

But the objection raised against these statistics is that the epidemic and institutional dysentery and that of war in temperate climates, are caused by the Shiga bacilli. In the bacillary dysentery of Japan, Shiga has never seen liver abscess as a complication. Asylum dysentery has been studied by Mott and Durham² in England and by Vedder and Duval³ in America. Even in one-half of the cases of dysentery due to Shiga's bacillus, Birt found multiple foci of suppuration in the liver. But Jäger has shown that epidemic dysentery may be of the amoebic type, as in the recent epidemic in the German army in 1901, in East Prussia. We may however draw the conclusion that bacterial dysentery is not followed by tropical abscess of the liver.

¹ Dr. Abercrombie's *Diseases of the Stomach*, second ed. Page 266.

² Mott. *Transac. Epidemiolog. Soc.* 1902.

³ *Journ. of Exp. Medicine* vi, p. 181.

There are other observers who can trace liver abscess as a complication of dysentery in the autopsy.

Observer.	Year.	post mortem of cases of dysentery.	Locality.	No. of liver abscess.	Per cent.
Schneider	1873	395		57	14
Beringer-Ferand	1883	411	Senegal	143	35
Annesley		29	India	21	72.2
Haspel		25		13	52
Gluck		28	Bucharest	16	57
Morehead		30	Bombay	12	40
Cheyne ¹	1818	30	Dublin	4	13.3
Parkes		25		7	28
Waring		300	India	96	31
Woodward		3680		779	21
Sanitary Commissioners	1888-94	465	India		35
Ranking		127		28	22.05

Ranking, out of a total of 150 autopsies, found:—

Dysentery with hepatic abscess = 41

„ without „ „ = 99

Hepatic abscess without dysentery = 10

150

¹ Dr. Cheyne in Dublin Hospital Reports Vol. III.

The table compiled by Hirsch¹ gives a ratio of 19·2 cases of concurrent abscess of the liver in every hundred fatal cases of dysentery :—

Table of the frequency of hepatic abscess in cases of dysentery (from post mortem reports).

Authority.	Locality.	Total deaths from dysentery.	Concurrent abscess of liver.
Ranking	India	140	41
Waring	"	257	69
Ballingal	"	35	4
Eyre	Madras	118	27
Moore	Bombay	494	90
Stovell	"	129	25
Macpherson	Calcutta	293	46
Chakrabarti	"	39	3
Marshall	Ceylon	247	49
Arthur	Burmah	20	7
Taylor	"	53	8
Bourgarel	Cochin China	22	7
Gayme	"	13	6
Julien	"	108	8
Wilson	China	61	2
Bédié	Algiers	10	6
Catteloup	"	240	47
Letelier	Chili	46	10
Roulet	Rocheport	50	2
		2377	457

¹ Geog. and Hist. Pathology. Vol. III. p. 412.

The association of liver abscess with dysentery has been however observed by many authorities :—

Observer.	Country.	Cases of liver abscess.	Cases of dysentery.	Percentage of cases associated with dysentery.
Annesley	India	29	21	72·3
Waring	"	204	...	72·2
Sanitary Com- missioners (1896- 1900).	"	509	...	53·0
Rogers	Calcutta	63	...	90·48
Sachs	Egypt	48	...	41·7
Kartulis	"	500	...	55 to 60
Zancaro!	"	444	...	59·0
Haspel	Algeria	25	13	52·0
Kelsch and Kiener ¹	"	{ 500 314	...	85·0 75·0
Smith (1870-95)	Seaman's	45	38	84·0
"	Hospital.			
Waterman Ed- wards		699	...	72·1
Budd	"	17	10	59
Campbell (1870-89)	Guy's Hospital.	69	22	32

¹ Kelsch and Kiener. *Traite des Maladies des Pays Chouuds. Paris, 1889.*

Kelsch. *La. Sem. Med.* 1900 March 7. P. 80.

The following table shows the frequency of hepatic abscess in cases of dysentery:—

Observer.	Country.	Cases of dysentery.	Cases of liver disease.	Per cent.	Cases of liver abscess.	Per cent.
1	2	3	4	5	6	7
Macpherson	General Hospital, Calcutta.	160	84	52.5	21	13.1
French Surgeons	Algeria	157	65	41.4	20	12.7
Marston	Malta	48	4	8.3
Eyre	Madras Fusiliers.	118	49	41.6	27	22.8
Waring	India	633	149	23.5
Stovell	General Hospital, Bombay	129	71	55.0	25	19.3
Leith (1 year)	"	47	4	8.5
Do. (several years).	"	92	14	15.2
Moore	"	196	60	30.6	35	17.8
		1580			299	
Innes	Secunderabad	7	17.9
Parkes	Burma	25	7	28.8
Marshall	Ceylon	25	7	28.8
Macnamara	Madras	51	26	50.9
Morehead	Bombay	30	12	40.0
		131			52	
	TOTAL ...	1711			351	20

18 A

34 B

40 C.

Moore¹ points out that 8 observers out of 14 accord a percentage of 18 cases of liver abscess in 1580 cases of dysentery. He explains the high percentage in the cases grouped under C by the supposition that possibly all cases of liver diseases are included in the column 6 and not liver abscess alone. Again if liver abscess be a common complication of, and is caused by, dysentery, how are we to explain that 77 per cent. of cases of dysentery grouped under A developed no hepatic abscess. Azevedo Sodré remarks² that "if we take the figures furnished by the statistics of Waring, Moore, Macpherson, Marshall, Morehead and Dutrouleau, we shall have a total of 1,997 antopsies of dysentery with 407 abscesses of the liver" or 20 per cent only. The question however can be now answered by the fact that formerly amoebic and bacillary varieties of dysentery were not recognised, and that liver abscess occurs as a complication of amoebic dysentery only.

So the old statistic are useless for determining the true relation of hepatic abscess and dysentery, for then

¹ Diseases of India, 2nd Edition.

² Twentieth Century Practice of Medicine. Vol. XVI. P. 252.

amœbic dysentery was not identified. So let us study some of the recent statistics :—

	Observers.	No. of amœbic dysentery.	No. of liver abscess.	Country.	per-cent.
Second Observation.	Kruse	11	4	Philippine Islands United States. Egypt.	36·3
	Pasquale				
	Councilman	9	6		66·6
	Lafleur				
	Strong	96	14		14·6
	Osler ¹	100	25		25
	Griesenger	186	2		1·07
	Harris	35	2		5·7
	Fletcher ²	119	27		22·6
	Craig	74			33
	Councilman			Tropical	
	Lafleur ³	2590	507		20
	Musgrove				
	Strong	100	...		23
	Kruse				
	Pasquale	57	...		11
	Harris	95	...		15

Thus we see that the ratio of dysentery to liver abscess is by no means constant. In some cases it is as 4 to 1. But in others the ratio varies within wide limits. Again we know that liver abscess is rare amongst the natives in places where the foreigners suffer to a marked degree; and also it increases by war and other accidental causes.

¹ Osler, *Theory and Practice of Medicine*. P. 196. 1904.

² Fletcher, *Journ. Am. Med. Assoc.* Vol. xli. P. 430.

³ Amœbic Dysentery. *John Hopkin's Hosp. Rep.* 1890-91.

So the relation between abscess and dysentery is a contingent one; other factors determining whether dysentery will give rise to abscess or not.

The modern pathologists, however, look upon dysentery as the sole cause of tropical liver abscess.

Is dysentery
the sole cause?

Macleod in Shanghai found in 40 cases of liver abscess, history of previous dysentery in 39. Manson¹ supports the same view and asserts that in 75 per cent. of cases of hepatic abscess a history of dysentery can be elicited. Olmeta and Vaillard regard dysentery as the sole cause of liver abscess. The researches of Dutranbau and of Kelsch and Kiener "have shown the close relation which exists between dysentery and suppurative hepatitis. These two endemic diseases describe a parallel curve, and in all latitudes any influence, which like war, famine, etc., determines an increase of dysenteric troubles, will also cause an increase of hepatitis." Rogers in Calcutta found 90 per cent of his cases associated with dysentery. In 56·5 per cent. he had both clinical and post mortem proof, in 20·63 per cent. post mortem proof, and in 14·3 per cent. clinical proof only, of the occurrence of dysentery.

Macleod explains the absence of dysentery in the observations of others by the following remarks:—

¹ Manson. "Abscess of the Liver." Qain's Dict. of Medicine.

1. History of antecedent dysentery may be overlooked.

2. Extensive dysentery may not give rise to active subjective symptoms.

3. Many patients forget to mention dysentery in their history and often confound it with diarrhœa. So we cannot affirm that dysentery was absent in these cases.

But we have the evidence of other observers in the field:—

Observers.	No. of fatal liver abscess.	No. in which dysentery present.	Examination for amœba.
Morehead	21	0	
Kartulis	33	22	11 had no dysentery. No amœba in pus or abscess wall.
Kruse }	15	8	7 "
Pasquale }	5	1	4 "
Hong Kong, 1903			
Sanitary Commis- sioners, Bombay }	74	2 to 3 per cent.	72 "
1888-94.			
Childe	Many	0
Meyer (Medical lege, Bombay.)	"	0	No history and no post mortem signs of dysentery.
Davidson	"	0	
Frerichs	8	0	
Louis }	16	Ulceration in	2 of which are tuber-
Andral }		3	cular.

Even amœbic abscess has been found to occur in cases where the colon was normal at the autopsy. (Burton,

Flexner, Thompson and Fitcher). Kelsch and Nimier¹ deny that amœbæ are responsible for the production of liver abscess.

Eichhorst calls attention to the fact that abscess of the liver is especially a disease of the tropics independently of dysentery, and the frequency of its occurrence in temperate climates may be a coincidence. Again "a history of previous dysentery is not sufficient to stamp a liver abscess as amœbic, or even dysenteric, if there are neither lesions, in the bowel nor amœbæ in the pus or in the wall of the abscess" (Davidson). It is moreover possible for a large single abscess of the liver to follow a non-amœbic diarrhœa. Large single abscess has occurred in persons who have never been out of England. Bartholow² remarks: "In the valley of Mississippi and its tributaries where liver abscess is comparatively a common disease, it has been found to be associated with an affection of the rectum—"the disease proctitis"—"although having a dysenteric form, is not ordinary dysentery". Raleigh³ says: "But, I am also quite satisfied that dysentery as it occurs under the ordinary interpretation of the term in Bengal and affection of the liver are not in any way whatever connected with or

¹ Bull. Acad. de Méd. Paris. 1900 t. xliii. p. 237.

² Pepper's System of Medicine, Vol. II, page 1004.

³ Observations on Idiopathic Dysentery as it occurs in Europeans in Bengal, by W. Raleigh, Calcutta, 1842, Page 10.

dependent on each other, and that whereas hepatitis is comparatively a rare complaint in the lower provinces of this division of India, dysentery is the most frequent and most fatal disease of Bengal under all its forms".

Again one should be careful in diagnosing a case as dysentery simply from the presence of blood and mucus in the stool. Cantlie¹ observes: "An intestinal flux with blood and mucus may or may not be dignified by the name of dysentery. We are apt to regard the presence of blood and mucus in the stools as indications of a specific disease, namely dysentery; but it is possible that mere portal congestion, with changes in the radicles of the portal vein or the intestine, may cause a flux of blood and mucus of a temporary nature and of non-specific character. Certain it is that, with the presence of pus in the liver, a flux of the kind, and lasting for a few days only, is a common feature. Dysentery, as a specific ailment runs a definite course; but an intestinal flux, resembling dysenteric motions, may come and go, during the presence of pus in the liver, in a somewhat erratic fashion totally unlike true dysentery".

Murchison is of opinion that as the result of observations in Burma² a large number of abscesses of the

¹ Encyclopædia Medica, Vol. 7, Page 27-28.

² Observations of the Climate and Diseases of Burma. Edin. Med. and Surg. Journ. 1854, Page 245-47.

liver met with in tropical countries can not be ascribed to dysentery. There were cases of recovery without any symptom of dysentery before, during, or after the malady, and this statement is corroborated by Morehead ¹, Bristowe² Frerichs ³ Maclean ⁴ and others.

Again it is quite possible for an abscess of the liver to be associated with amoebic dysentery, and to be the result of the dysentery, but yet not caused by amœbæ. Haasler could not detect in the abscesses of the liver of the German troops in China, any amoeba but found cocci and bacteria of the colon group.

Sir R. Martin and others were of opinion that liver abscess was intimately related with disease of the cœcum; but they could not detect hepatic abscess in more than 22 cases out of 72 cases where the cœcum was affected. Moreover their observation has not been confirmed.

Now if the presence of amoeba be the pathognomic of tropical abscess, the history of dysentery need not be invariably present, for amoebic diarrhœa may give rise to liver abscess. Also amoeba in the intestinal canal may

Dysentery may
be an indirect
cause.

¹ Researches on Dis. of India 1856 II page 10.

² Path. Trans. 1858 IX page 250.

³ Dis. of Liver II p. 116.

⁴ Reynold's System of Med. III. page 324.

cause liver abscess without giving rise to diarrhoea and dysentery. Again a patient who suffered from amoebic dysentery years before, may develop abscess of the liver as the result of dysentery and yet not caused by an invasion of amoebæ. In other words an amoebic dysentery may be the indirect cause of an idiopathic abscess.

Abscess sometimes occurs in healthy individuals after they have left the tropics for some years.

Late abscess

There is a remote history of dysentery.

The abscesses are sterile and do not contain amoebæ. But late abscess might be the development of latent abscess, and might have been caused by latent dysentery. For cases are not uncommon where a small tropical abscess with thick organised wall remains quiescent for months or years. Sir James Paget¹ calls such an abscess "Residual abscess" or abscess formed in or about the residues of former inflammation.

Cantlie² has shown the occurrence of tropical abscess

Suprahepatic
Abscess.

"between the layers of suspensory ligament having as boundary the peritoneum circum-

ferentially, the liver below, and diaphragm". It is not caused by dysentery but by chill.

¹ On Residual Abscess. St. Barth. Hop. Rep. 1869. V. 73.

² Cantlie J. B.M.J. 1899, vol. II, P. 646.

Thus we see that we have not one form of tropical abscess, but several varieties of it:—

1. Amœbic abscess; due to amœbic dysentery or diarrhoea, active or latent. Common.

2. Bacterial abscess: due to bacterial dysentery, being caused by the fusion of multiple small pyæmic abscesses. Rare.

3. Idiopathic abscess: not caused by amœbæ. It is met with commonly, though its relative prevalence depends upon time and place.

4. Suprahepatic abscess: not caused by amœba. It is the extrahepatic form of the idiopathic abscess.

5. The late abscess: It is remotely related to dysentery, but in most cases idiopathic.

So at the present state of our knowledge we can not accept dysentery or amœba as the sole cause of tropical liver abscess. There are other factors which determine the formation of a liver abscess.

On this point, Davidson remarks that there is practically no difference between idiopathic and dysenteric abscesses. "Both are tropical, both affect the European man more than the European woman; those addicted to alcohol more than the abstainers, those subject to tropical

malaria than those who escape the infection; those exposed to the fatigues and chills more than those not subjected to these influences. Both varieties occupy the same site, have the same symptoms and present essentially the same lesion"¹. The walls of large abscesses in both are irregular, and lined with a grey ragged membrane of granulation tissue undergoing necrosis. Externally, we find a zone of granulation tissue and degenerated liver cells and connective tissue elements; and then a third zone of connective tissue and flattened liver cells by compression. In old abscess a fibrous capsule is present. The chief points of difference may be shown as follows:—

*Idiopathic.**Dysenteric.*

- | | |
|--|---|
| 1. It is the result of inflammatory process. | 1. Result of necrotic process. |
| 2. Polymorphonuclear infiltration of liver tissues | 2. Absence of leucocyte accumulation either in the necrotic tissue or in capillaries. |
| 3. Amœba absent; cause of suppuration—bacteria. | 3. Amœba present, but the bacteria also. |
| 4. Shape: one or more buff coloured or yellowish spot; rounded—if one focus of suppuration; irregular outline—if many foci of suppuration. | 4. Smallest abscesses are one to five mm. in diameter. Spherical or ovoid. |
| 5. On section:—pus issues freely. Pus less viscid in character. | 5. Abscesses do not empty completely on section. Pus viscid and tenacious. |

¹ Tropical abscess of the Liver in Albutt's System of Medicine. Vol. II. Part II.

*Idiopathic.**Dysenteric.*

- | | |
|------------------------------------|--|
| 6. Hepatic cells not much altered. | 6. Hepatic cells undergo fatty degeneration. |
| 7. May have limiting membrane. | 7. Extends by necrosis of liver cells. No limiting membrane. |
| 8. Duration shorter. 7 weeks. | 8. Duration longer. 19 weeks. |

There is a great divergence of opinions also as regards the sequence of the abscess and dysentery. Some maintain that dysentery manifests

Sequence of liver Abscess and dysentery. first and the abscess is its sequel; others hold the opposite view that dysentery occurs as the result of suppuration in the liver; while there is a third party who thinks that liver abscess and dysentery are but different manifestations of one morbid condition, reacting on each other but not directly related as cause and effect. Besides there is a fourth class of cases in which abscess and dysentery alternate with each other.

Observers.	No. of Cases.	Primary.	Secondary.	Simultaneous.
Annesley ¹	...	{ dysentery liver abscess	{ liver abscess dysentery	} "
Morshead	...	{ dysentery liver abscess.	{ liver abscess dysentery.	} "
Flexner	...	{ dysentery liver abscess.	{ liver abscess dysentery.	} "
Kelsch	...	{ dysentery liver abscess.	{ liver abscess dysentery.	} "
Davidson	34	{ 21 dysentery 4 liver abscess	{ 21 liver abscess. 4 dysentery	} 9
Rogers	40	dysentery	liver abscess	
De Castro	...	dysentery	liver abscess	

¹ Volume II page 199.

Observers.	No. of Cases.	Primary.	Secondary.	Simultaneous.
Budd ¹	...	dysentery	liver abscess	
Murchison	...	dysentery but not always so.		
Foiet	40	{ 12 dysentery 28 liver abscess	12 liver abscess 28 dysentery	
Sachs	48	{ 9 dysentery 28 abscess 11 abscess	9 abscess 28 no dysentery 11 dysentery	

In those cases, where the hepatitis preceded the dysentery, Annesley referred the inflammation of the bowel to a morbid quality of the bile secreted by the liver. He explained the exemption of the small intestines from, and the marked liability of the colon to the inflammatory process as due to the longer delay of the decomposing faecal matter over the prominent folds of the latter; but hepatitis has not been known to cause any change in the quality of the bile secreted.

"Gauducheau² reports that when trying to reproduce abscess of the liver in a dog by a portal injection of pus from a human hepatic abscess he brought about a fatal amœboid dysentery". And Dr. Balfour remarks: "This is a matter of considerable interest. Indeed, from the post mortem appearances in a case of multiple liver abscess

¹ Diseases of the Liver. Page 86. J. T. M. Vol. IX. P. 52.

² Gauducheau, A (January 15th 1905). On Experimental Reproduction of Amœbic Dysentery by Intravenous inoculation of Pus from a Hepatic Abscess. J. T. M. Vol. IX. P. 52.

which came under my notice, I suggested¹ that, in some instances, hepatic abscess may precede a dysenteric affection of the large bowel."²

In the S. N. P. Hospital we have often observed that cases of liver abscess, apparently without any association of dysentery, developed the disease about 15 to 20 days after the operations. And when dysentery supervened in this way, it invariably proved to be a fatal complication.

Annesley³ describes a form of dysentery called hepatic dysentery in which "the association of structural changes of the liver with disorganisation of the large bowels is constantly observed" and remarks that the vitiated biliary secretions are the chief agents in exciting inflammation in the gut. Finlayson⁴ has pointed out how hepatic abscess may lead to congestion and even ulceration of the colon. In our cases we never found dysentery before the formation of liver abscess, unless we count cases who had dysentery long before.

The nature of the infection: The upholders of the dysenteric theory are not however of the same opinion as to the nature of the infection of the liver.

¹ Balfour (November 21, 1903) A case of multiple Liver Abscess. *Lancet* II. 1425.

² Review—Tropical medicine, Balfour, P. 49.

³ On Dysentery in India; by James Annesley.

⁴ Glasgow Med. Journal, Feb. 1873.

1. Some say that the infective material can only be conveyed by way of the portal vein and that it is of the nature of emboli. Then multiple abscesses would seem the inevitable result, but it is considered as the peculiarity of the dysenteric lesion to give rise to single abscess.

2. Other observers try to explain the association of liver abscess and dysentery by adducing a chemical agency as the mode of infection of the liver.

3. And lastly, many authorities think the infective material to be parasites: amæba or bacteria.

The specific toxic agency of tropical abscess,—parasitic, chemical or embolic,—on reaching the liver may set up a focus or foci of pus formation, that is, the formation of single and multiple abscesses would be merely accidental.

Even those who think dysentery to be the primary disease, are not unanimous as to the stage when liver abscess occurs. of the disease, when liver abscess is commonly found. Harris and others state that this complication always arises during the acute period, but observations show that it may develop at any time, and certainly not uncommonly after all symptoms of dysentery have ceased, or indeed, sometimes before any intestinal ones have developed. Strong detected liver abscess in a case who suffered from dysentery five months before. Fletcher's statistics also show that it may appear

at any time of the disease, but in the majority, the abscess becomes evident in the first month after the onset of dysentery. Again on this point the natives differ from the Europeans as to the interval that elapses between the attack of dysentery and the occurrence of liver abscess. Generally the natives who give history of dysentery refer to a period long ago—10 to 12 years back or more; and therefore though there is a sequence of events, any casual relationship between the two diseases is not proved.

Those who adduce dysentery as the sole cause of liver abscess, try to attach too much importance to dysenteric lesions found in the autopsy.

Such lesions are often discovered even when there is a total absence of clinical proof of the presence of dysentery. The real value of such lesions becomes useless for the following reasons:—

1. We do not know what is the maximum interval of time that can be allowed to elapse between the periods of attacks of dysentery and liver abscess, so that one may still be regarded as the cause of the other. Thus the history of previous dysentery without any reference to a reasonable period, can not be taken as conclusive evidence of the dysenteric nature of the abscess. Josserand thinks that abscess is possible even after 10 years.¹

¹ Journal. De méd. July 25, 1898.

2. The lesion may be of dysentery cured long ago. "Gives history of dysentery" might be said of every native at some period of life. It has no value in proving dysentery as the cause of liver abscess.

3. The age of the scar of dysenteric ulcer can not be known with certainty.

4. The scar may be of other forms of ulcers.

5. Even the post mortem signs are wanting in a large number of cases notwithstanding the ascertain of many that they are present in 90 per cent of cases. The view that this absence of post-mortem evidence is due to imperfect observations, can not be upheld in every instance, and the same weapon may be used against those who look upon dysentery as the sole cause. In the Pathological Museum at Netly, there are 48 specimens of tropical abscess of the liver, of which in 34 the abscess was uncomplicated with any intestinal lesion (Maclean) and in 11 only, it was present as a complication.

Again even admitting dysentery as the cause of liver abscess, we have the following facts to explain.
 Peculiarity of race and sex.

1. Though liver abscess is common to the Europeans in the tropics, it is rare amongst the natives.

Race of Army	Year.	Percentage of abscess to total mortality.	Percentage of dysentery to total mortality.
Native	1894	0.6	4.7
European	1894	7.4	3.8

So the relative liability of the Europeans as compared to the natives of India is 95.2 to 4.8.

2. This disproportion is inspite of the fact that the natives suffer more from dysentery.

3. The European women rarely suffer from liver abscess, though they are quite as subject to dysentery as men. Children often suffer from dysentery, but they hardly ever suffer from liver abscess.

4. The rarity of liver abscess in temperate climates is well-known. The absence of amœbic dysentery cannot explain it for large single abscesses do sometimes occur in temperate regions.

5. The Mahomedans of Bengal do not suffer so much as the Hindus. In my cases the Mahomedans were only 5, the rest being Hindus—95. The numerical strength of the Hindus does not explain it wholly. The Mahomedans again eat more meat but they are not addicted to drinking to such an extent as the Hindus are.

Now let us study some of the reasons against the view.

Reasons against that amœbic dysentery is the sole cause of
dysentery. liver abscess in the tropics:—

1. In many cases neither amœba was found in the pus or wall of the abscess, nor lesions of dysentery detected in the post-mortem examinations. Even amœbic abscess of

the liver has been found to occur in cases where the colon was normal at the autopsy¹. In 5 out of 27 cases recorded by Fletcher², the patients did not notice any intestinal symptoms.

2. The natives suffer greatly from dysentery but the Europeans suffer from the liver abscess more than they do.

3. The value of dysenteric lesion is often uncertain and cannot be relied upon.

4. Opinions defer as to the true sequence of liver abscess and dysentery. Flexner³ thinks that even in amœbic dysentery, the intestinal lesion may be secondary to amœbic hepatic abscess.

5. Geographical distributions of the two diseases do not coincide. The English suffer from abscess more in India than in Jamaica where dysentery is equally frequent.

6. Dysentery prevails in high altitudes, but liver abscess becomes rare there, as in Peru.

7. "About one-twelfth only of those suffering from amœbic dysentery develop abscess. Something more than

¹ Burton, J. T.:—Proc. Philadelphia. Pathlog. Soc. Jan. 1899.

Flexner, S.:—Am. Jour. Med. Sc. May 1897.

Thompson:—Manchester Med. Students, Gaz. 1903. P. 146.

² Fletcher:—Jour. Am. Med. Assoc. vol. xii. P. 480. August 1903.

³ American Journal of Medical Science. Vol. cxiii. P. 553. May 1897.

the presence of amœba in the bowel is necessary to establish hepatic suppuration, otherwise the natives would not be comparatively immune" (Davidson).

8. Liver abscess and diarrhœa: The two diseases may coexist; and in such cases the diarrhœa may depend on the liver disease or it may precede the formation of liver abscess which sometimes occurs insidiously.

CHAPTER III.

*Relation of liver abscess to 1. alcohol, mode of life,
2. food, 3. malaria.*

Now the question may be raised that if amœbic dysentery is not the cause, what is the cause of tropical liver abscess? Let us argue as follows:—

1. Europeans at home do not suffer from liver abscess. The reason may be that the climate is temperate and that amœbic dysentery is rare there, the bacillary form being common.

2. They often suffer from the disease in the tropics, so the cause of liver abscess must be chiefly climatic or amœbic.

3 But the European women and children and the natives do not suffer from the disease, though they are equally subject to the baneful influence of the tropical climate. So climate can not be the only cause. We have also shown that amœba can not be regarded as the sole cause.

4. The European males differ from the European females and children and the natives in their indulgence in alcoholic drinks, stimulating food and exposure.

5. The natives suffer from amœbic dysentery more than the Europeans. But they do not suffer from liver abscess to

the same extent as the Europeans do. Immunity or acclimatisation can not explain this difference for when the natives take to European habits, they often suffer from liver abscess.

6. The natives suffer from abscess when they become intemperate. The rich natives suffer more than the poor, for the former can spend money for drinks. But opposite is the condition as regards the incidence of dysentery.

7. Macpherson explains the less common occurrence of hepatitis among the British troops in India during the recent years, especially in Bengal, through the improvement in the soldiers' mode of life, including his dietary. Bryson also assigns the same reason for the decrease in the frequency of cases of hepatitis among the troops on the West Coast of Africa, the soldiers' daily ration of rum being reduced to half.

Daniell reports that hepatitis has become more common among the natives on the West Coast of Africa since their adoption of the European dietary.

So we can not avoid the conclusion that alcohol has something at least to do in the causation of liver abscess.

And we have the support of many observers for this theory of the causation of liver abscess. Waring traces alcoholism in 67·5 per cent. of 40 cases of liver abscess. Cayley¹ says that

¹ Trans. Calcutta Med. Soc. 1880, Vol. I, page 95.

liver abscess is rare amongst the total abstainers. Harvey supports the statement. Sandwith has observed that in Egypt the Mahomedans do not suffer from liver abscess as they are not addicted to drinking. It is rare amongst the teetotallers. Conwel has found that the native domestics who acquire the European vices suffer from liver abscess. The natives of India also suffer when they become alcoholic; and this fact is so well known that it has become as a proverb that he who drinks wine will die of liver abscess. In Philipine Islands Strong¹ found that in 12 cases among private patients 8 were alcoholic. In my 100 cases, only in 20, history of alcoholism was absent.

Even those who look upon dysentery as the cause of the disease can not ignore alcohol as a cause altogether. Manson² says:—"Finally a considerable proportion of cases of hepatic abscess arise under unknown conditions. In such cases, however, it is usually found that there has been more or less indulgence in alcoholic drinks, or the liver has been taxed by excesses in the use of rich foods and condiments, or exposure to extreme degree of temperature has occurred". Bartholow³ also echoes the same view.

¹ Osler's System of Med. 1907, Vol. II, page 503.

² Tropical Disease.

³ Pepper's System of Medicine, Vol. II, page 1003.

"As the native population and females in tropical countries are not affected, there must be other influences to the action of which the high temperature contributes. The rich and highly seasoned food in which the Europeans indulge and the consumption of alcoholic drinks are doubtless responsible in a large measure for the occurrence of the malady in such excessive proportion amongst them." Macnamara¹ in explaining the occurrence of granular degeneration in the liver of dysenteric liver abscess, says: "I have already alluded to causes which I think produce the granular degeneration of the liver. Shortly I believe them to be exposure to the sun, the heat of the climate, and malarious influences; these operating on a class of cases who eat largely, and as a rule indulge very freely in alcoholic liquors, would suffice to bring about a state of chronic engorgement of the liver substance, aggravated by occasional attacks of temporary congestion, and as a result of this granular degeneration.

Ruschenburger in explaining the frequency of hepatic abscess in the soldiers and sailors remarks that "these latter differ from every other class of society in their manners, their customs, their diet and even in their exposure to causes of disease. Intemperance is looked on by them as a virtue

¹ On the Pathology of Hepatic Abscess. Indian Ann. Med. Sc. 1882-83, page 9.

than vice, consequently nine-tenths of them are habitual drunkards. The quantity of whisky allowed daily by Government is in my opinion, sufficient to distroy the best constitution in a very short period." Annesley says: "Among the various influences which more directly occasion the supervention of inflammatory action of the liver, there are few more energetic than the immoderate addiction to the use of spirituous liquors and intoxicating drinks, which may be so readily obtained by the European soldier in every part of India". Nicoll also traces the real cause of abscess of the liver among the soldiers in Travancore to the incredible intemperance of the men. "I must own," says Henderson, "I never witnessed such drunkenness as took place in the corps.....At one time no less than 300 gallons of the pernicious liquor (toddy) were consumed daily in our barracks containing 700 men." The effect was that hepatitis carried off many. Vauvray in Egypt has seen abscess of the liver commonly amongst the Greeks who are generally intemperate; and Sachs says: "Alcoholic drink constitutes in almost every case the direct etiological factor in the disease which is the subject of my observations." Davidson remarks: "Alcohol except in the strictest moderation is a hepatic poison. In hot climates it can not be indulged in freely for any length of time without

impairing the integrity of the liver;”¹ and again he writes² “I would only add as the result of my own observations that constant use of alcoholic liquors, short of intemperance, especially tippling between meals, powerfully disposes to liver absces.” Martin³ traces liver absces to “evident and adequate causes; such as intemperance—violent exercise in the sun—or sudden exposure to cold, when the body has been sometimes in a state of perspiration. Of 40 cases in which the habits were noted by Waring, 67·5 per cent. were intemperate. Johnson, Twining, Day, Milward, Parkes, Bontius, Heyman, v. Lant, Marshall, Saux, Allan, Brasac, Rouis, Monilluc, Olmeta, Tschudi and others concur in the opinion that alcohol is the important factor in the etiology of hepatitis.

Now how does alcohol cause abscess of the liver?

How does alcohol cause liver abscess? “Intemperate habits lead to a special liability in men to a hyperæmia and congestion of the liver tissue by which its resistive faculty to pathological influences is impaired. In these circumstances, pathological influences which in the healthier condition of the organ—such as we assume

¹ Albutt's System of Medicine, Vol. II, Part II, page 573.

² Ibid page 591.

³ The Influence of Tropical Climate on European Constitution, 1841.

to exist more generally in natives and European women and children—would have been successfully overcome, gain the upper hand and lead to suppurative disintegration of the organ.”¹ The reason why Europeans in the tropics suffer so much from liver abscess is thus explained by Francis² :—“Inhabitants of cold countries who need warmth, consume combustible substances in considerable quantity, substances which contain carbon. The lungs of such consumers are called upon to undergo great exertion. They are in full activity; and consequently like a limb which may attain almost Herculean proportions from excessive use or a heart which becomes hypertrophied as the result of the continuous effort to overcome obstruction, they acquire their maximum of development. The lungs of Europeans weigh from 42 to 45 ozs. The lungs of Bengalees on the other hand who are not so actively engaged are much lighter, as are the lungs of Europeans who have lived for sometime in a tropical climate and undergone less exertion than they would have done had they remained in Europe.”

Males.	weight of lungs.	weight of liver.
Europeans	45 oz.	50 oz (Tidy).
Punjabis	35 oz.	46 oz (Beli Ram).
Bengali	32 oz.	45 oz.

¹ Manson's Tropical Diseases, page 379.

² Lectures on Medicine. By Francis M. B., 1868, Calcutta.

³ Trans. of Calcutta Medical Congress, page 126.

Thus it would be seen that the Punjabis who are physically as strong as the Europeans show a difference of 10 oz. in the weight of the lungs, but only 5 or 6 oz in the weights of the liver. This is more marked in the case of Bengalees whose lungs weigh 13 oz. less than that of Europeans but the difference in the weights of the liver is 5 oz, and this is inspite of the diminution in the stature of the inhabitants of Bengal. If the size of the organ has any relation to its capacity for work, then comparatively speaking it may be said that the lungs have more work to do in the temperate, and the liver, in tropical climate. "Residents in hot climates do not require so much carbons in their food; and if they consume it to excess, the result is disease. The lungs not being able to burn it off, it finds its way to the liver, which organ *may* get rid of it through the friendly intervention of a bilious diarrhæa, or if unable to do this, it will become diseased. The most common form in which carbon is thus presented to the liver is alcohol, which, owing to the depressing nature of the climate in which we live, and to other causes, too many are apt to have free recourse to. The result of this may be immediate or remote, for alcohol can not be consumed in a hot climate with the same impunity as in a cold one; and mischief will inevitably ensue. There may be immediate inflammation of the liver or fatty degeneration

of the different organs.”¹ Davidson admits “that the liver has more work to perform” but “this additional work, arises from the side of the gastro-intestinal canal, not from the lung.”² In temperate climates, alcohol gives rise to cirrhosis of liver; but in India alcohol does not indeed, give rise to cirrhosis so often as in Europe, for the reason suggested by Maclean that “the drunkards in India find a shorter road to the grave” by the formation of suppuration in the liver.

But there are objections to the alcoholic theory also. Raye³ mentions liver abscess in a European female who never tasted liquor in life. Sarbadhikari and Mookerjee support him by other cases. Chamber operated a case of liver abscess in a boy of 12 only.

The history of alcoholism is often absent; but this may be partly due to the fact that people often deny their bad habits and no sign of alcoholism can be detected in the post-mortem examination.

Waring concludes: “The mode of life has a strong influence in the predisposition to the development of a tropical abscess of the liver. The abuse of alcoholic liquors appears to be of

¹ Lectures on Medicine. Francis, 1868.

² Albutts' system of Medicine. Vol. II. Part ii. P. 572.

³ Transaction of the Calcutta Medical Society. Vol. I. P. 96. 1880.

very great importance, and some authors have stated that alcoholic drink constitutes a very important factor in every case of tropical abscess of the liver. This is, however, probably too sweeping a statement, as there are without doubt cases which arise quite independently of alcohol. The number of cases of hepatitis has been said to have diminished during recent years among the soldiers in India and on the West Coast of Africa, and this diminution has been attributed to the more sober habits of the men and the smaller amount of alcohol consumed by them. It is quite possible, however, that this diminution has been partially brought about by improved sanitary conditions and surroundings."¹

Waring² sums up his view thus: "Now the question presents itself—How are we to account for this exemption in the native and for the frequency of Hepatic affections among European male adults in India? We have seen in a former section that children are very exempt (though not to so great a degree as the native) from this form of disease and we should be led to infer that those conditions or circumstances which cause the exemption in the one case may operate in the

¹ H. J. Waring, *M. S. Diseases of the Liver*. P. 86.

² E. J. Waring's "Statistical notes on some of the diseases of India." *Indian Annals of Medical Science*, 1855. P. 457.

other. In what respects then do the conditions of the native and the European child approximate? In several it may be replied, in simplicity of diet, in the light and unrestrained style of dress, and above all in abstinence from stimulating and particularly from alcoholic drinks. It has repeatedly been suggested that a very high range of temperature and exposure to the direct rays of the sun operate in the production of Hepatic disease in India, but the very fact of the native convict being so exempt from its invasion tends to disprove any such idea, as perhaps there is no one class of persons in India who are so constantly, day after day for years together, exposed, whilst working on the roads, to the full influence of the sun's rays. So likewise with respect to sudden and great alterations of temperature, another alleged cause of Hepatic disease. The Indian convict, perhaps more than any other class of persons, is exposed to these, often working for some hours in the full heat of the sun and before his return to Jail deluged with rain—and yet we see not above 5 in 10,000 are attacked with Hepatic disease.

The native of India and the European child in India, as before observed, closely resemble each other in three respects—1st lightness and an unrestrained style of clothing (thus allowing unchecked operation to the cutaneous function), 2nd in simplicity of diet, and 3rd in the practice

of temperance. It appears to me that in the direct ratio as a person in India departs from either of these conditions, espacially the third, temperance, so are the chances of his becoming affected with Hepatic disease, increased. Next to the native and the child, we see that women are exempt from Hepatic disease. They often depart from the above conditions, particularly as far as dress and temperance are concerned, but rarely if ever to so great an extent as the Officer and Private Soldier who for the most part lead a life as diametrically opposed to that of the child and native as it is well possible to conceive, and these are subject to Hepatitis in an extreme degree. Rigid temperance *per se* is, however, no absolute preservative against the invasion of disease of the liver in India, and this leads us to consider in the next place—

THE INFLUENCE OF INTEMPERANCE IN PRODUCING HEPATITIS.

Table XXI.

Showing the admissions and deaths from Hepatitis among Teetotallers, the temperate and the Intemperate in six European Regiments, serving in Madras, in 1849-50,

Class.	Strength of Class.	Admissions with Hepatitis.	Deaths from Hepatitis.	Per cent. of admissions to strength.	Percent. of Deaths to strength.	Percent. of Deaths to Admissions.
Teetotallers	450	26	...	5.777
Temperate	4,318	249	16	5.766	0.370	6.421
Intemperate	942	96	2	10.191	0.212	2.083

These figures are too small and the data too limited to permit us drawing conclusions of any weight or value. It is, however worthy of remark, that double the proportionate number were admitted of those who were known to be intemperate over those who were either teetotallers or persons of known temperate habits”

The theory of alcoholism not only explains the formation of liver abscess but also the frequent association of liver abscess with dysentery. The European suffers from dysentery and abscess of the liver in the tropics ; and both the diseases may be traced to the habit of alcoholism. Davidson says that the prevalence of dysentery “can not be ascribed to the less favourable sanitary conditions under which he lives, as compared with the natives, nor to the less care taken to secure pure drinking water. His liability is rather the effect of climate, giving rise to intestinal congestion accentuated by alcoholic indulgence and the use of a diet unsuited to his new conditions of life”.¹ But as Europeans who drink alcohol in cold climate do not get abscess of the liver, simply alcohol can not be regarded as the sole cause of tropical abscess of the liver.

Is the habitual ingestion of highly seasoned food a Food. predisposing cause of liver abscess? An excess of animal food and spiced curries no doubt cause

¹ Albutt's System of Medicine, Vol. II, part II, page 530.

derangement of the functions of the liver in the tropics. But the Mahomedans who live largely on meat rarely suffer and so do the rich Hindus who, as a rule, eat much and take little or no exercise. "Weakened digestion is one of the early effects of tropical heat upon the constitution of the European. When the stomach is overtaxed by imprudences in diet, gastric digestion is imperfectly performed, and the toxic products of abnormal fermentations, absorbed from the bowel, irritate the liver."¹

Formerly malarial hepatitis was regarded as the cause of liver abscess. Morehead says that, in malarious districts, abscess of the liver occurs commonly with remittent fever without any intestinal lesion. Haspel, Annesley, Dutroulau, Foiret, Olmeta and Pellissier supported the same view. Now the opinion is exploded and malaria is excluded altogether from the etiology of the disease. We must admit with Soyard, Jean and others that abscess of the liver does sometimes occur in a chronic hyperainic liver of malarial origin but there can be no doubt that malaria is not the proximate cause of the disease. The two diseases may coexist but they are not coterminous in their geographical distribution. Malaria is now the scourge of the native population of Bengal—

¹ Albutt's system of Medicine. Vol. II. Part II. P. 573.

many villages have been deserted to escape its ravages; but we do not know of a single case where malaria could be identified as the sole responsible agent. In the tropics, congestion of the liver and hepatitis, as a consequence of malaria, are comparatively rare among natives, who are nevertheless more subject to malarial infection than the European. This becomes evident from the following figures :—

	European Army.	Native Army.	Year.
Malarial fever ...	290·5	372·7	1801
Congestion of the liver and hepatitis ...	16·2	0·97	

Again malaria induces cirrhotic changes in the liver, and not changes of a retrograde character. Malarial pigmentation does not lead to necrosis of tissues, though excess of the pigment may block the lumen of the hepatic capillaries (Cantlie). "But what is worse the malarial theory is in conflict with facts; malaria is infrequent in Egypt and unknown in Chilli, but these countries are prolific in the production of abscess of the liver. Again Lower Bengal is undoubtedly more malarious than Madras, but abscess of the liver occurs more frequently in the latter than in the former presidency. In Algiers, abscess of the liver is common in Oran, and rare in Bona and Constantine, but malaria is known to bear a reversed relation to those countries. Bertulus' also admits that

endemic hepatitis may occur in non-malarious regions, and Hirsch points out that, although Pellissier thinks, the coexistence of malarious fever and abscess of the liver in Reunion as a proof of the malarial theory, really proves the other way, for abscess of the liver has been at all times indigenous in the colony, whereas malaria became endemic there in 1866." The disease is also unknown in the malarious localities in the temperate zone as "parts of France, the Tuscan Maremma, the Calabrian coast. the Southern basin of the Danube, and the swamps of the Mississippi," thus proving that malarial fever can not be regarded as the cause of the abscess of the liver.

The combined effects of heat, alcohol and excess at the table render the Europeans seventeen fold more liable than that of the natives to hepatitis, notwithstanding the remarkable liability of the latter to malarial infection. Strong¹ also says that in the Philippine Islands at least, malaria is generally not a predisposing factor". The truth however rests midway between these two opposite extreme views. There are cases on record where the malarial element can not be ignored.

1. Bell's (Hongkong) case. A young officer had persistent fever which resisted quinine. On exploration

¹ Osler's System of Medicine, Vol. I, page 514.

pus was detected in the enlarged liver. His blood contained malarial parasites. No history of dysentery.

2. A French soldier was admitted for liver abscess with fever; there was no history of diarrhoea or dysentery. His blood contained parasite of tropical tertian. The temperature continued high even after operation and became normal by the subsequent use of quinine. And Davidson comments that it is impossible to exclude the influence of the malarious virus in the causation of the abscess.

Gluck believes that liver is more prone to suppuration when predisposed by amyloid or cirrhotic change of malarial origin. We cannot accept malaria more than as a predisposing cause—

Year.	Hospital.	Total No. of cases.	No. of cases of Malaria.	No. of cases of Hepatitis.
1899	{ S. N. P.	16614	4296	157
	{ Presidency General	4111	576	84
1900	{ S. N. P.	20108	4925	215
	{ P. G.	3841	598	86
1901	{ S. N. P.	20212	3928	183
	{ P. G.	3719	405	86
1902	{ S. N. P.	21036	4918	127
	{ P. G.	4307	714	50
1903	{ S. N. P.	21016	3875	123
	{ P. G.	4673	639	59

The Presidency General Hospital and the Sambhu Nath Pandit Hospital are situated on the opposite sides of the same street. The former is for the treatment of Europeans and the latter for natives exclusively. Now if we compare the figures as shown above, we find that the total number of patients treated in the Sambhu Nath Pandit Hospital is 4 times, the number of cases of malaria, 7 times, but the number of cases of liver diseases is two times only greater than the cases treated in the Presidency General Hospital, year after year. This shows that natives though more liable to malaria than Europeans are less liable than them to liver diseases in general.

Again hepatic abscess is known to occur where malaria is absent as in Seychelles Group and the island of Rodrigues.

CHAPTER IV.

Relation of Liver Abscess to stimulants. Personal factors—Race, Age, Sex, Temperament, Constitution, Acclimatisation.

Undue stimulation of the liver may lead to inflammation of the organ. In this way the habitual use of tobacco and coffee is looked upon as a cause of the disease. Excessive smoking, though decidedly less injurious than alcohol, has likewise a detrimental influence on the stomach and liver (Davidson).¹ Browser associates hepatic abscess with excessive tobacco smoking.² I knew a patient who died of liver abscess due to the excessive smoking of *Gudi*. Opium-eaters do not suffer from the disease.

Race. We have seen that natives are less liable to liver abscess than Europeans. It will be quite apparent if we compare the rate of admission for abscess amongst the European and the native in India :—

Observer.	Locality.	Years.	Ratio of admission for hepatitis among British and native regiments.
Balfour ...	Madras ...	1829-1838 ...	120 to 1.
Cornish ...	„ ...	1842-1859 ...	100 to 2·1.
Morehead...	Bombay	100 to 2.

¹ Davidson: Albutt's System of medicine 1st Ed. Vol. IV, page 137.

² Trans. Calcutta Med. Soc. Vol. I. 1800, page 96.

As regards the death rate from liver abscess amongst the European and the native soldiers, it was higher in the latter formerly :—

Sepoys	9.1	per cent. of the whole number.
British Soldiers	4.6	„ „ „
European Hospital, Bombay	14.1	„ „ „
Native	„	„	34	„ „ „

But now we find a higher death rate amongst Europeans :—

Year.	Race.	Death rate per 1000 from liver abscess.
1889	{ European army	1.24
	{ Native army	.05
1890	{ European army	1.05
	{ Native army	.03
1891-1900	{ European army	1.25
	{ Native army	0.06
1900-1903	Jail population	0.09

Haspel has observed that the foreign legions—the Italians and Spaniards in Algeria,—are less subject to hepatitis than the natives of the Northern Europe. Strong has seen only one case in a native of the Philippine Islands.

The comparative immunity of the native of India from the liver abscess can be best explained by their temperate habits and light diet. Even in India the Hindus supply the bulk of the cases of liver abscess—the Mahomedans, though numerically less than Hindus, suffer much less in proportion to their number.

			Cases of Dysentery.	Abscess.
Natives	79,723	127 or 1 in 628.
Europeans	7972	441 or 1 in 18.

Sachs also found "among the non-Mahomedan residents of Cairo, who may number some 20,000, a not inconsiderable series of cases, in nearly all of which the principal cause has been the free use of spirituous liquors." "Of the two women in my list," says he, "one was a true votary in the service of Bacchus and Venus; and of the men, the larger number were heavy brandy-drinkers, some were rots, and only a few were temperate enough to restrict themselves, apart from their regular wine at meals, to small portions of liquor now and then." Henderson however found it common among the Mahomedans in India "who were permitted by religious prescriptions to live luxuriously" and these were either the rich who observed luxury at the table or the drunkard.

Is there then any racial peculiarity to account for the greater liability of Europeans than the natives to abscesses of the liver in the tropics?

The question must be answered in the negative, for "the rates of cases among the British troops in India is greatest in the lower ranks of the service, the malady being rare among the officers; and the same fact presents itself on the West Coast of Africa and in the West Indies, where it

is again the common soldiers and the labouring class that suffer most; also in Egypt, where the disease is oftenest met with among working people, Greeks especially." The strangers in a tropical climate are no doubt apt to develop hyperæmia of the liver but its extreme rarity in the female sex, including women who have come to the tropics from other countries and are subject to the same climatic influences as the men (Hirsch) can not be explained by racial peculiarities.

Bryden says that the liver abscess is a disease of degeneration. It is most commonly met
 (b) *Age.* with in the adult life. The association of liver abscess with dysentery was noticed often in old men by the French surgeons in Algeria.

Observer.		Age.
Manson After childhood; common—20 to 40
Rouis ¹ 12 to 75 years.
Common age in soldiers	...	23 to 27 "
" " in civil life	...	30 to 45 "
Bartholow 11 to 54 "
Rolleston (16 cases)	...	Children; all with dysentery.
Murchison } Fayrer } 20 to 45 years.
Hongkong Hospital Returns	... 3 years	} No amœba found.
" " " 1903...	10 "	
Soldier's children in India ² —38,305—of which 3 died of liver abscess=0·08 per 1000.		

¹ Rouis', *Traite pratique des maladies des pays chaud.*

² Buchanan, *Journ. of Trop. Med.* Vol. I, page 173, 1899.

General Hospital Calcutta (Birch)...20-59 years.

Duckworth ... Rare under 20 and frequent after 25.

J. Waring¹ ... Never in children; rarely old men.

Poiret ... 20 to 25 years.

Amberg saw 12 cases in children,² but amœba was present in one only. Moncorvo has seen a fatal case in a child aged 2 years;³ Arnot⁴ reports a case in a child 2½ years and Finzio, in a child 5 years old.⁵ Cheevers⁶ collected only three cases: in a child of 17 months, in a European child of 3 years and 4 months; and in a Parsee child of 10 years.

In the European army in India. Waring's time:—

Of 227 cases

10	=16 to 20
59	=21 to 25
53	=26 to 30
42	=31 to 35
35	=36 to 40
13	=41 to 45
9	=46 to 50
2	=51 to 55
3	=56 to 70
1	=72

112 „ bet. 20 to 30; 48.4 per cent.

Youngest=15 years in a girl.

Oldest=72 years.

¹ Diseases of the Liver, page 86.

² John Hopkins Hosp. Bull. Dec., 1901.

³ Rev. Mensuelle des Malad de lenfawe, Dec. 1899.

⁴ B. M. J. 1903. I. page 189.

⁵ La Padiatria 1896, page 310.

⁶ Choever's. Commentary on The Diseases of India, P. 616.

Macpherson :—

Uncommon = under 20 years.

Most common = 25 to 35 „

Most fatal = 30 to 35 „

IN THE EUROPEAN ARMY IN INDIAN, 1901-03.

4 Cases under 20 years.

10 „ between 20-25.

22 „ „ 30-35.

18 „ „ 35-40.

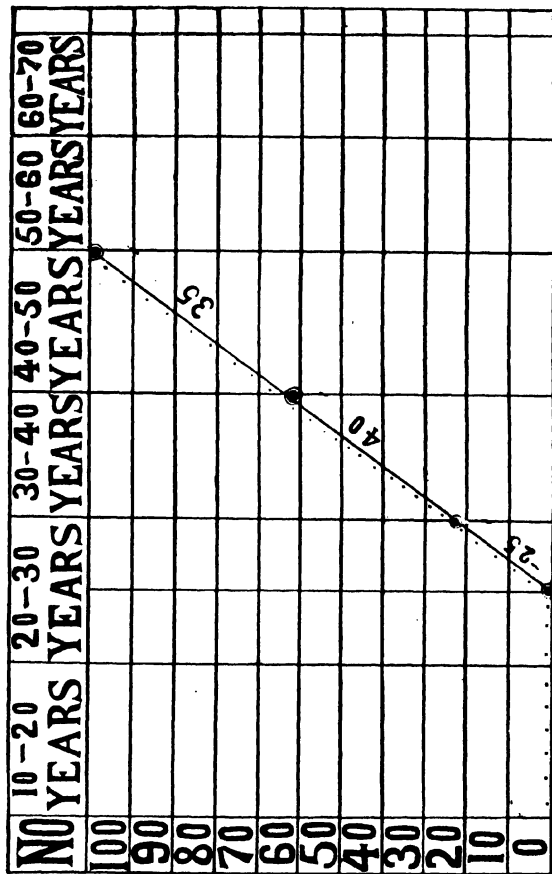
26 „ „ 40 and upwards.

The cases of which I have notes occurred between the ages of 30 to 50 years. I have never seen a case in the children amongst the natives. Waring, Macpherson, Morehead and Cheevers agree that “that childhood, which is especially liable to dysentery, enjoys an almost complete immunity from idiopathic hepatic abscess.”

The females suffer rarely from liver abscess. This is especially true of the natives of India, (c) Sex. where I have never seen a native female falling a victim to liver abscess. This is also true of Europeans for during the 20 years ending 1860, nearly 6½ per cent. of all the deaths in soldiers were caused by liver disease, but one per cent. of all the deaths in soldiers' wives.¹

Observers.	No. of Cases.	Male.	Female.	Proportion.
Rolleston ...	13	11	2	} 30 : 1
Rouis ...	256	250	8	
Bartholow ...	12	11	1	
Waring ...	300	291	9 { 3 Native 6 European	

12. Chart of ages of 100 Cases.



To face page 84.

Observers.	No. of Cases.	Male.	Female.	Proportion.
Indian Statistics				
	Mortality (1901-03)	{ 1.5 per. 0.24 per 1,000 1,000		7: 1
Birch	...	18	13	5
De Castro ¹	...	170	162	8
Zancarol ²	...	157	145	12

Futcher reports 3 cases in the female sex. Strong has not seen a case in a woman in the Philippine Islands. Carter states that he has seen 3 cases in Filipinos.

Now the question why the females are immune from liver abscess though they suffer equally with the males from amœbic dysentery can be easily answered by the theory of alcoholism. They do not suffer because—

1. They are temperate in habits
2. They are less exposed to chills and do not undergo excessive fatigue.

3. Menstruation acts as a safety valve which lessens the liability to hepatic congestion. (Van der Burg.)

Rouis finds that a combination of the lymphatic and

(d) Tempera- nervous temperament seems most favourable to the malady. Those who suffer from the gall-stones, also show a predilection to the liver abscess; but in such cases the calculi, by their arrest in the duct,

¹ De Castro "Des abcès du foie des pays chauds, et de leur traitement chirurgical." Paris 1870.

² Zancarol, "Traitement chirurgical des abcès du foie des pays chauds." Paris 1893.

may ulcerate through, producing peritonitis and adhesions, and eventually abscess of the liver. Thus the element of temperament is only secondary.

(e) Constitution. The disease is common in persons of strong constitution.

Observers.	Cases.	Previous health.
Saux 52 47 (Good.)
Rouis 116 102 ..

Ranking concludes from a number of observations
(f) Acclimatisation. "that the risk of death by hepatic abscess is greatest in the first five years of service ; that the risks decrease during the second period of five years, rise again in the third, and in the fourth at the minimum." Brydon's figures show that long residence increases the liability.¹

Length of service does not in fact, as Waring states, confer a great immunity from suppuration of the liver. Surgeon Major Web died in the 27th year of his service.

Macpherson: 1851-54. European troops in Bengal:

8 died under 1 year.	
63 ..	1 to 3 ..
55 ..	3 to 5 ..
45 ..	5 to 7 ..
57 ..	7 to 10 ..
32 ..	10 to 14 ..
2 ..	14 to 20 ..
0 ..	above 20.

¹ See Albutt's System of Med. First Ed. Vol. iv, page 139.

In Algeria, the French Surgeons have noticed that men who have been long in the country are often the victims of liver abscess.

The earliest years of residence in the tropics no doubt, show a higher percentage,—about 40 in the first three years, but the older residents are by no means exempt.

Davidson proves this by the following table:—

1901-1903.

	Under 1 year.	1-2	2-3	3-4	4-5	5-10	10 and upwards
Percentage of mortality from liver abscess ...	18	7	10	12	14	25	14
Invaliding from hepatitis and liver abscess ...	3	10	8	10	11	24	34
Occurrence of liver abscess (Waring) in 114 cases ...	22	10·5	10·5	[50 per cent. occurred in people who had been less than 4 years in India.]			

Similarly there is no acclimatisation for dysentery with the older residents, as will be seen from the table of Bryden—

	1st year.	2nd year.	1-5 years.	5-7 years.	7-10 years.	10 and upwards.
Percentage of mortality from dysentery ...	9·6	10·4	9·0	10·1	13·3	13·7

CHAPTER V.

Pathogenesis of liver abscess. Relation of liver abscess to various forms of dysentery and ulcers. Path of the inflammatory process.—Bile ducts, Portal vessel, Peritoneal cavity. Causes of the association of liver abscess and dysentery: a reflex action, a common cause, concurrence of causes.

We have discussed at length the relation of hepatic abscess to climatic dysentery, malaria and alcoholism. We have found that, at the present state of our knowledge, it is difficult to identify a single cause of the disease. The different theories of its causation have supporters and opponents. I am inclined to the view that hepatic abscess has not such a single cause but it depends upon a combination of causes. There is a large amount of truth in each of these observations—but not the whole truth. A theory comprising these theories would explain the phenomena more satisfactorily.

Fayrer observes¹: “The great disproportion of the occurrences in different parts of the world, where the same climatic conditions appear to exist, seems to suggest that it cannot be attributed to mere heat, malaria, moisture

¹ Hygiene and Diseases of Warm Climates. Davidson. Art. Tropical Liver Abscess by Sir Joseph Fayrer.

or sudden alterations of temperature. It must be due rather to the combined effects of these with varying food, habits and modes of life, and the different degree of strain imposed thereby on the functions of the liver."

It has been repeatedly observed that any of the causes mentioned before, cannot set up abscess of the liver. Inhabitants of the tropical climate do not often suffer; nor do the alcoholics of the temperate climate. Amœbic dysentery does not always develop the hepatic abscess as a complication, and the natives who are saturated with the parasites of malaria rarely seek medical aid for abscess of the liver. The liver, which is physiologically healthy, can withstand these causes separately; but becomes inadequate for the struggle when two or more causes act in concert. Thus amœbic dysentery will easily help the amœbic invasion of a liver, which is in a state of degeneration by a tropical climate and the habitual ingestion of alcoholic stimulants. But even if the patient be temperate, the amœbic toxins may be of sufficient virulence to break down the physiological resistance to microbic invasion. Again the functional activity of the liver might be deranged by a chill, and hepatitis may supervene and ere long the degenerated areas will be invaded by the amœba or pyogenetic bacteria. This hypothesis is quite simple and attractive; and it explains the various anomalies in the etiology of the disease satisfactorily.

And we have the sanction of an authority, for Davidson takes the same view of the matter.

Now let us study, How does dysentery cause liver abscess? Ewald remarks that "the liver forms a sort of catchpit between the right side of the heart and the blood of the portal vein, which receives all the toxic matters absorbed from the bowel and either holds it and gives it up little by little to the blood, or destroys it, or excretes it again with the bile into intestine". In chronic dysentery which is generally the result of amœbic invasion, the amount of toxic matter is increased. The liver is inefficient to counteract the toxic influence and so abscess results.

But the question may be asked, Why do not bacillary dysentery, typhoid and tubercular ulcerations give rise to hepatitis or abscess?

Bacterial dysentery generally runs an acute course. To set up degenerative and inflammatory processes it requires some time, for the toxins formed are soon eliminated by the stool.

Macleod suggests that the typhoid and the tubercular ulcers are surface lesions unattended with abscess formation in the walls of the gut. There is also free escape of the germs of putrefaction ; but in dysentery, besides the surface

lesion, there is really abscess formation with burrowing and retention of pus beneath the mucous membrane, and there is great liability to invasion of micro organisms into the radicles of the portal vein. According to this view, the liver abscess becomes a pyæmic process. But the dysentery preceding abscess of the liver is often of a catarrhal form, and not so severe as is required in the explanation. It can not explain the fact that dysentery, though more common amongst natives than Europeans in India, is seldom followed by liver abscess in the former. Neither can it solve the question of single and multiple abscesses, unless their formation be regarded as accidental.

Martin thought that liver abscess was intimately connected with lesions in the cæcum, but of 72 cases where the caecum was observed, liver abscess was found in 22.

Again abscess is not uncommon after other ulcers of the intestine. Kartulis reported six cases of liver abscess with amœbic appendicitis. In three the intestinal lesions were severe in the caecum and the appendix. In one the appendix was most extensively diseased while in the caecum only small ulcers existed. He suggests that the portal of entry of infection may sometimes be through lesions in the appendix. Rolleston reports a case;¹ and Cassuto

¹ Rolleston, Diseases of Liver 1906, P. 125.

detected typhoid bacilli in the hepatic pus¹. Hölschner² found 12 cases of liver abscess in 2000 cases of typhoid fever.

Fayrer cites two examples³ in the Pathological Museum, Calcutta Medical College, which "illustrate the formation of pyæmic abscess in the liver, in one case as the result of amputation of the leg, in the other from injury to the bladder and rectum."

Brussais, who regarded inflammation of the intestinal canal as the common cause of inflammation of the liver, thought that the irritation from the mucous membrane of the intestine, especially duodenum was transmitted along the (a) Bile ducts, bile ducts to the liver. Andral and other French physicians supported this view. But in most cases such anatomical continuity of propagation can not be traced; and again in such a supposition, hepatitis ought to be more frequent than it really is.

Ribes first brought forward the theory that the inflammatory process from the intestinal mucous (b) Portal vessels, membrane is transmitted, through the medium of the *veins* to the liver.

¹ Cassuto: These de Paris, 1900.

² München, med. Wochenschrift 1891, No. 4, 5.

³ Davidson Diseases of the Liver P. 645.

Budd adopted this theory in a still wider sense that the portal blood becomes contaminated with morbid matter from the various kinds of ulcers of the gastro-intestinal mucous membrane and that this being arrested in the degenerated liver substance commences the abscess.

But in such cases there is absence of inflammation in the portal vessels which is required by the theory. Cambay never found intestinal pylephlebitis in his cases.

This objection however is not valid for the morbid matter arrested in the degenerated liver substance, does not cause abscess by setting up inflammation, but by destroying the nutrition of the part and thus causing its death. Amœbæ are frequently found in the veins of the submucosa and in the portal capillaries.

Macnamara says: "I suspect Dr. Budd's doctrine may be in many cases true, so far as it holds the abscess to be due to the poisonous effects of morbid matter taken up by the portal vein and precipitated upon the liver". But in Budd's theory "it has been far too generally assumed, that the dysentery precedes the hepatic inflammation; almost all physicians who have acquired their experience in warm climates have arrived at the conviction, that the dysentery is often preceded by the hepatitis, or that the two may be developed simultaneously, and that hepatitis frequently

occurs without any disease of the intestine." (Frerichs).
 "And may it not be that the liver abscess (or the state of the liver which precedes it) so impairs the functional activity of the organ that the large intestine is compelled to take on some kind of compensatory action, and is thereby either predisposed to the attack of the dysenteric poison (if such there be) or, as an immediate result of unwonted overaction, becomes the subject of dysenteric changes?"

The other method of transmission is through the
 (c) Peritoneal cavity. Councilman, Lafleur
 and Rogers support this theory. The
 parasites migrate through the intestinal wall and then
 invade the liver. Lafleur detected amoeba over the
 peritoneal surface of the intestine and liver in a case with
 peritonitis.

As to the cause of the association of liver abscess with
 dysentery, Macnamara questions that in
 many cases, Is not the reflex action exerted
 upon the liver by the diseased intestinal
 mucous membrane the cause of the congestion of the liver,
 which accompanies the first stage of
 dysentery? "And may it not be that one
 effect of this congestion upon the degenerated structure of
 the liver is so to interfere with its nutrition as to bring
 about a local death at one or more points?"

The cause of
 association of
 liver abscess
 with dysentery.

(a) One a reflex
 of the other.

Then again it may be contested that both the diseases are the effects of a common cause and
 (b) Both de- depend on the same cause. hence the co-existence of the two diseases in the same individual. And may we not point out alcohol as the cause, in tropical climate, of so much degeneration, not only in the liver but also in the mucous membrane of the intestine as to make the degenerated areas of the two organs fit soils for amœbic infection simultaneously, and which might have produced in certain persons either of the diseases separately?

Or perhaps with more reason, we may look upon the two diseases as depending on a concurrence
 (c) Both de- depend on a concurrence of causes. of causes which individually will cause only one of the diseases. This view has the support of Murchison ¹ who says that "supposing, for example what is properly the truth, that dysentery is the result of a poison inhaled or swallowed in drinking water, and that hepatitis may be caused by a chill in a person whose liver has been congested by a residence in a hot climate, aided by intemperance, irritating ingesta, and exposure to the malaria of tropical fevers, it is readily conceivable that in a country like India where these causes so often operate simultaneously, attacks of dysentery

¹ Murchison's Diseases of the Liver. 2nd Ed. 1877, page 181.

and hepatitis—combined as well as separate—should not be uncommon.” So Fayrer says¹: “Dysentery, malarial fever, hepatites and liver abscess may co-exist with or supervene on each other, as results of climatic influences.”

See Davison. Diseases, of the Warm climate P. 644.

CHAPTER VI.

Morbid Anatomy—Macroscopic and Microscopic appearances. The initial lesion,—cells, vessels, connective tissue. Formation of tropical abscess. Single and multiple abscesses. Their relations to dysentery. Site of the abscess. Why is the right lobe the common seat of the abscess? Its form and size. Quantity of pus. Contents. The liver pus. Is liver pus sterile? The organisms found in it.

The macroscopic appearance of the liver found in a
Morbid Ana- case of hepatic abscess in the post-mortem
tomy. examination :—

“ As sequel to dysentery, we find in the liver two kinds
of lesions, firstly local necrosis of the
(a) Macroscopic appearance. parenchyma, scattered throughout the organ
and possibly due to the action of the chemical products of
amœba, and secondly abscess, single or multiple. When
single, the usual seat is the right lobe either towards the
convex surface near its diaphragmatic attachment, or on
the concave surface on the proximity of the bowel.
Multiple abscess are small and generally superficial. In
early stage the abscesses are grayish yellow, with sharply
defined cantour, and contain a spongy necrotic matter, with
more or less fluid in its interstices. The larger abscesses
have ragged necrotic walls, and contain more or less viscid,

greenish-yellow or reddish-yellow purulent material mixed with blood and shreds of liver tissue. The old abscesses have a fibrous wall of a dense, almost cartilaginous toughness."

The state of liver outside the zone of the abscess is variously described by the authorities :—

Rouis :—Volume of the organ is normal in 63 per cent. of his cases. The vessels may increase or decrease. Of 25 cases he found the liver diseased in 19 and healthy in 6.

Waring :—The liver is softened or altered in colour.

Kelsch and Keiner :—Parenchyma of the liver outside the abscess zone is as a rule healthy.

Davidson :—Liver is rarely healthy after chronic dysentery.

The inter-lobular areas are always the first to become disintegrated and the periportal areas persist until detached by the necrotic process. The microscopic changes in the liver cells consist of a granular degeneration; and I have seen it often with the deposition of intercellular granular matter. In the earlier stages the cell wall and nucleus may be detected but often cannot be made out. The cells often look scaly and their edges ragged and disintegrated and often look like cell walls filled with granular debris. Fat-

(b) Microscopic
Appearance.

globules are often present and the cells may show extreme fatty degeneration.

The edge of the abscess consists of necrotic liver cells where amœba, leucocytes, blood corpuscles and fibrin filaments are found. The capillaries are dilated, filled with blood and often contain the parasite. Outside this layer is a zone of connective tissue cell-proliferation in which mononuclear small cells are abundant; but the hepatic cells are compressed or atrophied. This layer is surrounded by a hyperæmic zone in which thrombi may be found in the branches of the portal veins, and the amœba and bacteria abound. The absence of leucocytic infiltration should be noted as it usually accompanies suppuration of bacterial origin.

The contents of the abscess cavity are composed of fatty and granular detritus, degenerated hepatic cells, pus globules and amœba which are commonly found in the abscess walls chiefly in the necrotic zone. Mallory has devised a differential stain, by which the amœbæ can be identified in the tissues. Cultures often give negative results,—they are usually sterile.

Opinions vary as regards the initial lesion in hepatic Initial Lesion. abscess. Virchow¹ describes the lesion beginning in the *cells*, which become granular, then opaque, finally soften and suppurate. Klebs on the other hand

maintain that the changes in the cells are due to compression exerted by the mass of septic micrococci distending the adjacent *vessels*, and then the process of suppuration commences on the portal side of the lobules. There is a third opinion—elaborated chiefly by Leibermeister and Koster—that the initial lesion is in the *connective tissue*.

The tropical abscess of the liver forms thus:—
The first stage is the occurrence of hyperæmia of the
Formation of organ. The vessels dilate due to the deposit
the tropical
abscess. of the morbid matter. Gradually, the cells
become cloudy, granular and opaque. A yellow spot soon
appears, surrounded by translucent greyish ring and here
suppuration begins. The cells disintegrate and a purulent
collection is formed. The size of the abscess depends upon
the state of the liver. The abscess may be encapsuled
by a limiting membrane, which is developed from the
connective tissue becoming the seat of the inflammatory
process.

In chronic cases, the abscess walls may be firm and thick, but as a rule the limiting membrane is wanting, and section of the wall shows an internal layer grayish in colour, shreddy, consisting of necrotic hepatic tissue, pus, cells and amœba, a middle layer brownish red in colour, and an external zone of hypermænic liver tissue.

If it be not confined by a limiting membrane, it

enlarges at the expense of the liver substance, and though it becomes encysted, after a quiescence of variable duration, begins active efforts to communicate externally.

The tropical liver abscess, though commonly, is by no means, single. The following table will show the number of single and multiple abscesses as observed by the different surgeons :—

Observer.	Locality.	No. of cases.	No. of single abscess.	No. of double abscess.	No. of multiple abscess.
Waring	India	268	177 or 61.5 p. c.	33 or 11 p. c.	<div> <div>11 Triple,</div> <div>17 Quadruple, 5.6</div> <div>10 from 5 to 10</div> <div>40 more than 10</div> </div> <div> <div>36 p. c.</div> <div>"</div> <div>"</div> <div>"</div> </div> <div> <div>78 or 27</div> <div>p. c.</div> </div>
Niblock	Madras General Hospital	83	"	"	17 "
Meyer	Bombay	100	"	"	"
Rouis	"	75	"	11	14 "
Davidson	"	"	"	"	"
Sanitary Commissioners, India 1896-1901	India	509	34	"	66 "
Zancarel		562	<div>Single</div> <div>Multiple</div>	<div>=</div> <div>3 : 2</div>	
Moore		28	11 or 39 p. c.	6.21	11 or 39
Futcher		18	10 " 55.5 "	"	8 " 44.4
Strong	Philippine Island	23	13 " 56.5 "	"	10 " 44.4
Craig		24	9 " 37.5 "	"	15 " 62.5
Rogers		32	21 " 65.6 "	"	11 " 34.1
Rollleston	St. George's Hospital	13	10	"	3

It will be seen that there is a large preponderance of single abscess in the statistics of all the observers except in the case of the Sanitary Commissioners; single abscesses would be eliminated by operation, while the multiple cases would come prominently in the autopsy. This may explain the preponderance of the multiple abscesses. The single abscess though frequently found in dysenteric cases, is more commonly met with when there is no intestinal lesion. But it should be remembered that in many cases, single abscess results from the coalescence of two or more abscess. This is evident from the fact that the cavity is often loculated indicating that several original foci of suppuration have united together.

The following table (Dividson) represents diagrammatically an analysis of the Indian results, shewing the relation of a single and multiple abscess to intestinal ulceration; it merits some attention on account of its bearing on the question of the existence of dysenteric and non-dysenteric form of liver abscess :—

Multiple		Single		No ulcer.		Ulceration	
198	73 p. c.	271	27 p. c.	98	57 p. c.	171	43 p. c.
		Ulceration	53 p. c.			Single	24 p. c.
		No ulcer.	47 p. c.			Multiple	66 p. c.
509 cases				509 cases			
Multiple		Single		No ulcer.		Ulceration	
140	59 p. c.	238	41 p. c.	140	1 p. c.	388	59 p. c.

Abscess occurs more frequently in the right lobe than the left; the upper and posterior part of the right lobe is the common seat. Much less frequently, it occupies the concave surface of the liver.

Observer.	No. of cases.	Right lobe.	Left lobe.	Both lobes.	Lobus Spigelli.
Waring	300	163 or 67.3 p. c.	16 or 6.6 p. c.	35 or 14.4 p. c.	
Davidson		70 to 80	5 to 15		2 to 5 p. c.
Maclean		9	9		
Rouis	639	78.6	16.8		4.6
Bortholow		70			
	132	85	45		2
Haspel		30	1		

Now the question may be asked, Why is the right lobe

Why is right lobe often attacked? more liable to the disease than the left? This can be explained if we consider—

- (1) The right lobe is six times larger than the left.

(2) Serege's ¹ experiment proves that blood from the intestines is chiefly directed to the right lobe.

(3) Rogers thinks that as amoebic dysentery invades the ascending colon and hepatic flexure, there is possibility, in cases of superficial abscess at least, of the direct passage of amœba across the peritoneum to the right lobe.

The hepatic abscess is generally round in shape; but
 Form. it often becomes irregular and even digitate and loculated when the abscess results from the fusion of multiple foci of suppuration. The smallest abscesses were often sphericial or ovoid.

The size of the abscess depends upon its age, the
 Size. stage of progress and the resistance of tissues around it.

The quantity of pus contained in the cavity may vary
 Quantity of from 2 to 3 oz to 150 oz. The largest
 pus. sized abscess may contain 3 to 6 litres of pus. In one of Annesley's cases the quantity reached 90 oz. In Inman's case, it measured 13 pints. Fayrer² removed 80 oz and again 50 oz of pus from the same liver within a week. Forty to sixty

¹ Soc. Med. des Hop. April 26, 1901. Journ. de Med de Bordeaux. May 25, June 1 and 8, 1902.

² See Davidson Tropical Disease. P. 643.

ounces of pus is the quantity that I found in the majority of my patients. Single abscesses are generally large and may vary in size from an orange to a cocoanut. In Lieutund's case, the quantity of pus was 12 pounds. The large abscesses often involve more than three-fourth of the entire organ; and in rare cases the whole liver may be converted into a huge abscess sac. Strong reports such as a case, the liver and abscess weighed 3700 grams. Portal, Haspel and others record cases in which the entire organ was converted into an enormous bag of pus.

Multiple abscesses are often small, varying in size from a plum to an orange. According to Lafleur, the smallest visible abscesses have a diameter of 1 to 5 mm.

The contents of the abscess depend upon the nature of Contents. the disease, the rate of its progress, the state of the hepatic parenchyma and the presence and absence of a limiting membrane. In chronic cases where the abscess has a dense limiting membrane, the pus is laudable, or dry and cheesy; in the embolic cases, it is dark brown, ichorous or grumous; in the true tropical cases, the contents may be creamy, sanguinolent, or sero-sanguinolent, dark or grayish or dirty white and purulent, viscid or watery, frequently like thick gruel, chocolate coloured, sticked with bile or blood, closely resembling 'anchovy-sauce' and emitting a sickly, disagreeable,

mawkish odour. This is the so-called hepatic pus, characteristic of the tropical abscess. It is sometimes tinged green by bile (Rokitansky) and becomes offensive if the abscess be situated near the colon. In one instance it had the sour smell of chyme, though no connection with the stomach was found. Fagge mentions a case, in whom the pus had a peculiar nauseous odour and a brick dust colour.

The hepatic pus consists of fatty liver cells, fat globules, debris of hepatic tissue, leucocytes, red blood corpuscles and frequently shreds of necrotic liver tissue. In it, are often found the hæmatoidin and Charcot-Leyden crystals, bile sometimes, oil-globules, bacteria and amœba. The polymorphonuclear leucocytes are either absent or present in small numbers only.

As regards the presence of bacteria and amœba opinions differ :—

Observer.	No. of Cases.	Organism.	No. of cases in which they occur.	Remarks.
Rogers	84	Cocci	16	In 6 accidental contamination is possible.
Kartulis	19	Bacteria free or included in amœba.	13	
Kruse } Pasquale } Morgenroth (China)	6	" Streptococci and Staphylococci	5	Amœba often absent in pus or abscess wall.
Fletcher	27	Bacteria	15	
Strong	23	"	13	

In many cases the pus is sterile. Cantlie says: "The liver abscesses arising from metastatic or pyæmic infection will be found to contain bacteria in every case; but that idiopathic or true tropical abscesses, the result of hepatitis may not show bacteria in their contents until after the pus finds exit from the liver. But the formation of pus independently of bacterial infection would transgress the basis of our pathological knowledge." So this is explained as due to the fact that the bacteria present in the early stages, die out later on. Manson explains that the absence of the amœba in the first liver pus is due to the death of the infusorian amongst the dead central pus; and he says the amœba is only to be met with in newly formed pus which finds ready escape, or in scraping from the walls of the abscess cavity. Calmette's theory suggests that the sterile abscesses are not caused by any pyogenic bacteria but by some chemical substance derived from the bowel and thus explains the absence of bacteria in the abscess cavity. This theory has the support of Grawetz and Scheuerlen who artificially produced liver abscess by injection of cadaverin and peptrescine. Cantlie hints that the liver pus may be necrosed liver tissue floating in a sero-sanguinolent fluid, and is not like ordinary pus, so bacteria need not be present in it. Councilman and Lafleur, however, in addition to the abscess

Is the liver pus
sterile?

formation, have also described a wide spread necrosis of the cells round the central veins of the lobules. They suggest that this is due to soluble chemical products of the amoeba. But the evidence is not conclusive.

The various kinds of organism found in the hepatic pus are :—

Bacteria, Staphylococci, Streptococci, Colon Bacillus Organisms. and Bacillus Pyocyaneus. These are abundant in the capillaries between the pyogenetic membrane and the liver substance.

Amoeba is found in pus or abscess wall. They are abundant in the smaller abscess in the pyogenetic membrane and also in the thrombi of the portal vein.

Crimm and Berndt found active flagella in the pus ; and Manson came across a ciliated infusorium like Balantidium Coli. The peculiar organism I found in the pus and also in the stool of a patient will be described later.

Netter found that after ligature of the bile duct, staphylococons aureas appeared in the gall-bladder ; thence they penetrated into the liver and the blood, and abscesses were produced in the liver and other organs. Kartulis found amoeboid organisms in dysenteric liver abscess, but not in the idiopathic variety, in which he found Bacillus pyogenes fætidus and albus. Even in the dysenteric variety, he thinks

that the dysenteric liver abscess is due to pyogenic bacteria, carried thither by amœba from the intestines. Macfayden¹ made a bacteriological examination of a case of tropical abscess of the liver, and found that the staphylococcus pyogenes aureus undoubtedly produced the suppuration.

Bonsfield² records a case of liver abscess, in the pus from which, a diplococcus was found soon after operation, simulating the gonococcus in appearance and staining reaction.

¹ Macfanydens "Notes on the Bacteriology of Tropical Abscess of the Liver in Davidson's Diseases of Warm Climates pp. 658—661.]

² Bonsfield. Lancet January 1908. A case of liver abscess due to a Diplococcus similar in appearance and staining reaction to the Gonococcus." Journ. of the Royal Army Medical Corps Vol. X. No. p. 80.

CHAPTER VII.

Cause of Suppuration—bacteria and amœba. How do amœbæ act? History of amœba. Their various forms. Biology of Amœba. Distribution of amœba in the body. Its presence in healthy persons. Behaviour towards physical and chemical substances. Characteristics of amœba coli and amœba hystolytica.

What is the cause of suppuration—Bacteria or Amœba?

In the pus of hepatic abscess both amœba and bacteria are present; so it becomes difficult to decide which of them is the real cause of suppuration. The experiments performed by Musgrave and Clegg are useless in proving either way as they were made with amœba grown with bacteria. When the vena portæ is the channel of infection, amœba possibly carry bacteria with them. But we cannot even assert so far, when the infection occurs directly through the peritoneum. At the present stage of our knowledge we may think that amœbæ with or without bacteria set up dysenteric liver abscess.

HOW DO AMŒBÆ ACT?

1. Councilman and Lafleur think that the soluble products of amœba give rise first to necrosis of hepatic cells

and the breaking down of the tissue is caused by active penetrating and disintegrating movements of the organism. The amoeba works in the living tissue around the abscess. This theory has the support of Marshall¹, Manson² and Rogers.

2. Another opinion is that the amoeba is simply the carrier of bacteria to the hepatic tissue; and the suppurative process is the work of the bacteria (Kartulis). But it is unsubstantiated and implies the presence of the amoeba in the liver abscess invariably—an assumption without the corroborative testimony of facts.

3. Kruse and Pasquale regard both the amoeba and bacteria to be responsible for the abscess formation.

4. Miss Sheldon Amos³ thinks that the amoebic infection is a secondary one; and that the amoeba gains a footing in the liver when it has been damaged by bacterial invasion. Kelsch and Nimier deny that amoeba is responsible for the production of liver abscess.⁴ Cantlie says: "The amoeba plays no part in the changes which lead up to an abscess, and it is only when the abscess is tapped and flowing that the amoeba travels towards the region."⁵

¹ B. M. G. June 10th, 1899.

² Tropical Medicine page 294.

³ Journ. Path. and Bacteriology, viii. P. 346. 1912.

⁴ Bull. Acad. de Méd. Paris 1900 t. xliii. P. 237.

⁵ Encyclopædia Medica, vol. 7. P. 22.

Lambl in 1860 first found amœba in the stool. History of Lewis in 1870 found them in the stool of amœba. a cholera patient in India. Loesch found numerous motile amœbæ about 5 to 8 times the size of red blood corpuseles in the stool of a man suffering from chronic diarrhœa. He injected the stool containing amœba into the dogs, one of which shewed dysenteric lesion in the intestine. Grassi detected amœba in the stool of the healthy persons and so did Cunningham and Lewis who thought them to be the transitional stages of flagellates and sporozoa. In 1883 Koch in Egypt found them in the deeper parts of ulcer in cases of dysentery, and hinted at the casual relationship to the disease. Kärtulis next noted a relation between the number of the organisms and the severity of the symptoms of dysentery. Other observers, Osler Councilman, Lafleur, Simon, Musser and Stengel in the United States confirmed the identity of the amœba, and Normand, Sonsino, Perroncito and Halva supported the amœbic theory of dysentery by injection experiments. Calandrucio swallowed encysted amœbæ which re-appeared in his normal stool. Massiutin detected amœba in intestinal diseases other than dysentery. Cohen, Nasse and Quincke and Roos (Germany), Kovacs (Austria) and Lutz (Brazil) confirmed the observations of Kärtulis. Kärtulis produced dysenteric lesion with the cultivated

amœba and with the pus from a liver abscess containing amœbæ but no bacteria.

Quinke and Roos found two varieties of amœbæ one agreeing with Lösch's *Amœba Coli* and the other showing a different encysted state and failing to produce dysentery by the injection experiments and therefore called *Amœba Coli Mitis*. He distinguished a third harmless variety—an inhabitant of normal intestine—the *Amœba Vulgaris*. Schuberg, Kruse and Pasquale also found amœba to be the normal inhabitant of intestine sometimes, and succeeded with the pus (bacteria being absent) of hepatic abscess in producing dysentery in cats. They recognised a pathogenetic *amœba dysentery* from non-pathogenomic *amœba coli*. Zancarol showed that pus from hepatic abscess in which amœba could not be found, set up dysentery in cats, thus proving that sterility can not be decided by the absence of amœba.

Celli and Fiocca have described six different kinds of amœbæ found in the human stool:

1. *Amœba lobosa* var. *guttula*.
2. „ „ „ *oblonga*.
3. „ *spinosa*.
4. „ *dliaphana*.
5. „ *vermicularis*.
6. „ *reticularis*.

Boas obtained negative results by the injection experiment in some cases, but it can be concluded that amœba is the essential factor in the causation of amœbic dysentery. Harris succeeded in producing dysentery by injection of fæces containing amœba and in one case amœbic abscess of the liver. Attempt to produce dysentery or abscess by the injection of amœba-free bacterial culture of dysenteric stool failed in all cases. Marchoux succeeded in producing not only dysentery by injection of dysenteric stool in cats but also single and multiple abscesses of liver. Thus we see that amœbi dysentery has a claim to be accepted as the cause of tropical abscess of the liver. But we must remember that amœbic dysentery as a distinct disease is not established beyond doubt. Cantlie says¹ :—"The very presence of the amœba coli is now known not to bear a pathological significance, for it is a resident in the healthy gut. The normal presence of the amœba in other organs has not been fully investigated; but in many abdominal lesions the amœba finds its way thither, perhaps as a consequence of the pathological change rather than as a factor in the origin of the changes."

Biology of Amœba. The amœbæ are classed under the

¹ Encyclopædia Medica. vol. 7, P, 21

Rhizopoda of the Protozoa. They are unicellular parasites, Morphology. having a granular endosarc and a clear ectosarc. Those organisms move by means of pseudopodia. It possesses a nucleus placed eccentrically in the endosarc. A nucleolus is also seen in coloured preparations. Red blood corpuscles, pigment granules and bacteria have been observed in the endosarc.

In preparations from the stools, Borrell's blue is Stain. the most satisfactory stain. In tissues the theonin stain, and for staining permanent preparations from cultures, Wright's modification of Romanowski's method are recommended.

All attempts to obtain culture of pathogenetic Biology. species by Musgrave and Cleg, and other pathologists have failed. The saprophytic amoebæ have been grown on solid culture media free from all extraneous substance except bacteria by Ogata, Celli, Fiocca and others. It has been shown that certain amoebæ develop only in relation to bacterial symbiosis. Tsujitani obtained cultures of amoebæ with cholera spirillum. Musgrave and Cleg obtained plate cultures of amoebæ from dysenteric stool and liver abscess in man. They were able to separate bacteria from amoeba by heat; but in no case did they observe multiplication of amoebæ in the absence of living bacteria. In the amoebic abscess of the liver where

bacteria can not be found, they consider it possible that other organic substances, possibly of a labile nature may in the living body act in the same way as the bacteria. The selection of the bacterial partner is very difficult especially of the pathogenetic amœba which would not act injuriously, for instance in the monkeys without its pathogenetic bacteria.

Cassagrandi and Barbagallo have studied the development of non-pathogenetic species; and Schaudinn has given an exact account of the pathogenetic species which is called *Entamœba histolytica* in contradistinction to the first variety or *Entamœba coli*.

Distribution of Amœbæ in the Body. "Besides the intestine and neighbouring tissues, the abdominal cavity, abscess of the liver, lung, and pleura, amœbæ have been found in the following pathological conditions: in ascetic fluid in cases of abdominal tumor; in necrosis of the jaw-bone; in abscess of the mouth; and in disturbances of the bladder and urine. Celli and Fiocca reported their cultivation from the larynx and lungs in cases of tuberculosis, and Gross and Sternberg found them in tartar scraped from the teeth".¹

¹ Osler's System of Medicine. Vol. I. P. 494.

Many observers have noted the presence of amcebæ in healthy persons:—

Observer.	Year.	Number of persons.	Purgative used.	Amcebæ found in the stools.	Country.
Schuberg	... 1893	20	Carlsbad	10	...
Kruse	} ... 1894	38	...	38	...
Pasquale	
Strong	} ... 1899	...	Rochelle Salt	4 per cent,	Phillipine Islands.
Musgrave	
Schaudin	...	68	...	34	East Prussia
	...	385	...	256	Austria
	$\frac{1}{2}$ of cases	Berlin
Craig	... 1905	200	...	65	...
Dock	Nil	U. States
Zorn	Nil	Munich
Musgrave	300	...	101	Manila
				61 had dysentry.	
				$40 \left\{ \begin{array}{l} 8 \text{ died.} \\ 32 \text{ developed} \\ \text{dysentery.} \end{array} \right.$	

The presence of amœba in healthy persons does not prevent us from saying "that they are not suffering with an early or latent form of the disease (dysentery), or that the malady does not exist in its incubation period, unless we follow them over long periods of time in which no disease develops."¹ Amœbæ also occur in the stools of those suffering with other diseases than dysentery as chronic diarrhœa, cholera, intestinal tuberculosis, typhoid fever, hæmorrhoids, etc.

¹ Osler's System of Medicine. Vol. II, p. 498.

Behaviour of the amoebæ towards physical conditions :

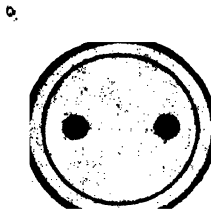
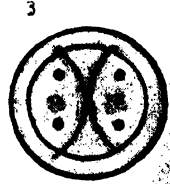
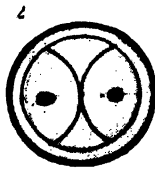
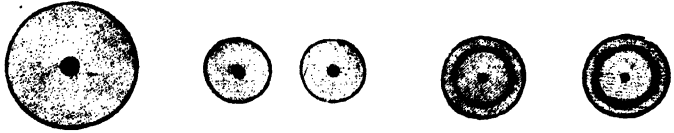
Observer.	Amœba.	Temperature.	Effect.
Musgrave Clegg	} Stools or culture ...	-75° F.	No loss of motility
Many		"	Loose there motility.
Craig		Freezing	Instant death.
Tuttle	} Dysenteric Freshwater	70° F.	Fatal.
		high or low	Motile.
Kruse Pasquale	} Dysenteric	Frozen & thawed...	Produced dysentery after injection.
Harris		0° C.	Not fatal.
Musgrave	Encysted culture of dysenteric.	-12' C. (for 45 days)	A transplant gave fresh growth, but failed in the next experi- ment.

Behaviour of amœba towards chemical substances :

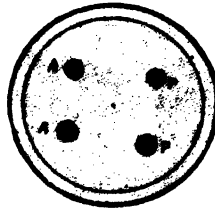
Observers	Amœba in.	Chemical agent	Strength	Time	Effect on amœba.	Below 70° F
Harris	Stool	Quinine Sulph	Saturated solution	...	Nil	...
		Boric Acid		
		Quinine Bisulph	1 to 300	10 min.	Death.	...
		Hydrogen Dioxide	Weak solution.	...	Death.	...
		Pot. Permanganas		
Rogers	Wall of liver abscess	Toluidine blue	Dilute Acids	Hours.	No death.	...
		Quinine Sulph.			Motility lost.	...
Tuttle	Bichloride of mercury	1 to 500	5-15 min.	Death.	...
		Silver Nitrate	1 to 100	...	Checks motility	...
		Saline solution	1 to 10,000	Hours	No death.	Death.
		Hydrogen Peroxide	1 to 100	5 per cent.	Encysted.	
Musgrave Clegg.	Slant culture of amœba with bacteria.	Quinine Hydrochlor	5 per cent.	15 vol.	No death.	...
			1 to 2,500		Encysted.	...
			5 to 8 min.	10 min.	Broken up.	...
		Acetozone, 1 per cent. acid 1 to 5,000 } to Phenolphthalein.	1 to 2,000		No amœba in first inoculation but bacteria grew out.	...
					Death.	...

Observer.	Amoeba in.	Chemical agent.	Strength.	Time.	Effect on amoeba.
Thomas	Culture with symbiotic cholera spirilla	Boric-Acid	...		Nil
		Eucalyptol			
		Ichthyol			
		Cassia oil	1 to 1000 1 to 2000 1 to 4000	30 min.	Moderate.
		Quassia infusion			
		Tannic Acid			
		Copper Sulph.	1 to 1000 1 to 1000 1 to 1000		Cholera spirilla Death.
		Pot. Permanganate			
		Quinine Sulph			
		Alphozone	1 to 2000 1 to 500 1 to 2000	30 min.	Marked
		Pot. Permanganas			
		Quinine Sulph.			
		Silver Nitrate	1 to 500 1 to 500 1 to 2,5000		Most marked but no effect on cholera spirilla.
		Argyrol			
		Protargol			
		Thymol			

13. Life cycle of Amoeba



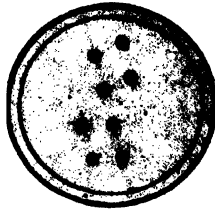
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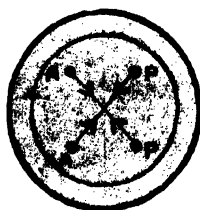
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1. *Amoeba coli*. 2. Binary fission. 3. Encysted stage.
 6. Part of it cast out or absorbed. 7. Formation of a new nucleus.
 10. Remaining nucleus again divides. 11. Again one-half absorbed
 into a single mass. 13. Active and passive pro-nuclei. 14. Their
 17. Free amoebæ.

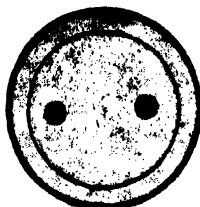
coli. Diagramatic



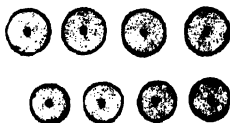
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14



15



17

4. Daughter Nuclei, and division of protoplasm. 5. Chromidia
8. Nucleus divides into two. 9. One-half absorbed or cast out.
or cast out. 12. Thickening of the wall and fusion of protoplasm
fusion. 15. Zygote nuclei or Sankarya. 16. Cyst of 8 nuclei.

The life cycle of *Amœba Coli* and *Amœba Hystolytica* according to Schaudinn :—

Amœba Coli : Multiplies by binary and also by multiple fission, to form 8 free amœbæ which feed and grow. So they vary between 8 and 50 μ in diameter. This fission is more marked in the fluid stool. Then the amœba becomes encysted and passes out of the gut. Each cyst contains a single uninucleate amœba; the nucleus dividing, form two daughter neuclei which go to the opposite poles of the cyst. The protoplasm then divides; each nucleus now becomes resolved into chromidia, some of which are absorbed or cast out. From the remaining chromidia a new nucleus is formed, which divides into two, the one-half being absorbed or cast out. The remaining nucleus divides again, and again one-half is cast off. The cyst walls become thicker, and the protoplasm shrinks, the two distinct portions fusing into a single mass, in which are seen the two mature generative nuclei. Each nucleus divides into a pair of daughter nuclei—the active and passive pro-nuclei. The active pro-nuclei of each pair fuse with the passive pro-nuclei of the other pair; so the cyst now contains two zygote nuclei or synkarya. Each synkarya divides into four so the cyst contains 8 nuclei. At this stage, cyst requires to be swallowed up by a host; then the contents of

the cyst escape in the large intestine of the host and the protoplasm divides up round 8 nuclei to form 8 small amœbæ which just as in schizogeny, start on the vegetative phase and then complete the life cycle.

Amœba Hystolytica: Multiplies both by binary fission and irregular gemmation in which, multiple. Life cycle of *A. hystolytica*. nuclear division leads to the formation of infinite number of independent individuals. The average size varies between 25 to 30 μ in diameter, the nucleus being 4 to 6 μ in diameter. In unfavourable conditions, the formation begins of resisting phases, not however of cysts, but of spores; the nucleus first gives off chromidia and is then rejected. The chromidia collect near the surface, and ectoplasm grows out into little elevations, each containing numerous chromidia, which separate from the parent, as spheres of protoplasm to become afterwards spores. The remainder of amœba degenerates. The spores then become surrounded by an envelope. These spores can produce dysentery in feeding experiment; but nothing further in their life history is known.

Vedder¹ classifies the characteristics of *Amœba histolytica* and *Entamœba coli* as follows:—

¹ Vedder, E. B. (March 24th 1906). Journal American Medical Association.

ENTAMOEBA HISTOLYTICA
(DYSENTERIÆ.)**ENTAMOEBA COLI.**

25-30 Microns (not a distinguishing feature)	Size	10-20 Microns.
Usually some other shape	Shape	Spherical when resting.
Greenish	Colour	Opaque grayish.
Ectoplasm and Entoplasm easily distinguished	Protoplasm	Ectoplasm and entoplasm distinguished with difficulty.
Ectoplasm very refractive		Ectoplasm not refractive.
Ectoplasm finely granular		Ectoplasm homogeneous.
Entoplasm coarsely granular		Entoplasm finely granular.
	Pseudopodia.	
Large and easily distinguished		Hard to distinguish.
Certain ectoplasm and entoplasm		Entirely ectoplasm.
Many	Vacuoles	Often absent.
		Never more than one.
Often absent. When present its structure hidden except in stained specimens. Nuclear membrane not well defined. Changes position markedly.	Nucleus	Almost invariable, with well-defined nuclear membrane and other structure. In moving, organism retains relative position.
Many	Red corpuscles ingested.	None observed.
Great progressive motility	Motility	Often absent, or, when present, of limited extent and short duration.

Lösch, quoted by Manson, gives very similar characteristics, and in addition mentions:

Multiplication.

In the intestine by fission and budding. On hard faeces on outside the body resistant spores formed without encystment.	In the intestine by binary fission and also by multiple fission into 8 amœbulæ. On hard faeces and outside the body encystment and formation of 8 amœbulæ.
(These develop when swallowed)	(These are set free when swallowed)

[Quoted in Balfour and Archibald's Review—Tropical Medicine &c. Supplement to the Third Report of Wellcome Research Laboratories at Khartum 1908.]

For the cultivation of *amœba hystolytica* the following media may be used :—

1. The ordinary culture media, or a combination of tap-water 90, alkaline nutrient broth 10 and agar 0.5.

2. Hay or straw media: take 30 grams of hay or straw and 1 litre of water. Boil for half-an hour. Make the filtrate alkaline with sodium carbonate 5 grains. Boil and filter. By adding half per cent. of agar before neutralisation a solid medium can be obtained.

3. *Fucus crispus* medium: take broth or tap-water, add 5 to 15 per cent. of *fucus crispus*. Boil. Add 1 cc. of decinormal potash to 10 cc. of the medium.

4. To cultivate *amœba* with bacteria, prepare a bacterial medium composed of agar 20 grams, sodium chloride and extract of beef each .3 to .5 grams, with a final reaction of 1 per cent. alkaline to phenolphthalein (Musgrave).

5. Ordinary gelatine medium: wash the gelatine with distilled water and then sterilise (Lesage).

It is difficult to obtain a pure culture for there must be present bacteria as food of the *amœba*. If the *amœbic* cyst be treated for 72 hours with 20 per cent. solution of water-free soda, the bacteria die out. The *amœbic* cysts do not then grow on ordinary culture media but readily do

so if the cultures of pathogenetic bacteria be added. Thus the pathogenetic bacteria of a particular species of amœbæ may be determined.

In a paper read at the British Medical Association Meeting, Major A. R. S. Anderson, I.M.S., has shown that during the year 1905, 2359 cases of dysentery were admitted to the Port Blair hospitals, and of 920 of these the stools were examined microscopically. In 262 flagellates alone were found, 29 exhibited amœbæ alone, 456 flagellates and amœbæ, 4 flagellates, amœbæ, and *balantidium coli*, 2 flagellates and *balantidium* and 1 *balantidium* alone. In 167 no protozoa could be discovered. The flagellates consisted of *lamblia intestinalis*, *trichomonas intestinalis*, and *trichomonas hominis*. The last species was most frequently encountered and constituted about 75 per cent. of the total. As control the stools obtained by administering a watery purge of 210 patients admitted for disease other than those of the bowel were examined. Of these 7 exhibited amœbæ alone, 54 amœbæ and flagellates, and 80 flagellates alone. The larger number of the amœbæ found in the cases of dysentery were no doubt those described as *entamoeba histolytica*. In the non-dysenteric stools the amœbæ, but for the absence of blood corpuscles from their interior, could be in no way distinguished microscopically from *entamoeba histolytica*. The amoeba was charged

with causing dysentery because it was found in the base and surrounding tissue of dysenteric ulcers, but he thought that the same charge could with greater cogency be levelled against *trichomonas hominis*, in so much as it was found in the same situation and occurred more frequently than the *amœbæ* in the dysenteric intestine. In his 920 cases while *amœba* was found 488 times, *trichomonas hominis* was found 540 times. Since then 1 per cent. of all cases of dysentery, or 2 per cent. of cases of dysentery with intestinal *amœbiasis*, he considered that the *amœbæ* could not be regarded as the cause of abscess of the liver. That *amœbæ* were found in the interior of abscesses of the liver was undeniable, but it was a very weak argument that they cause the disease. Many observers had found flagellates accompanying the *amœbæ* in abscess of the liver, and yet while the *amœba* was incriminated the flagellate was exonerated. He had attempted the experimental infection of animals (one kitten, one puppy, and two monkeys) with dysenteric stools containing both *amœbæ* and *trichomonas hominis* administered by the mouth or rectum, but he had failed to produce either dysentery or abscess of the liver; notwithstanding that the introduced protozoa previously absent had persisted for many weeks in the stools of the experimental animals. In Dacca, and other places in Eastern Bengal he had found *entamœba histolytica* to be almost,

though not quite, as prevalent in cases of dysentery as in Port Blair and equally dissociated from abscess of liver. He believed, therefore, that *entamoeba histolytica* was not an important factor in the causation of abscess of the liver."

Before finishing the consideration of *amoeba* we would like to draw the attention of the medical profession to a peculiar kind of *amoeba* detected in the stool of a patient in the Darbhanga Hospital. It was a fatal case of dysentery in which the liver was enlarged and tender. We examined the stools and found innumerable organisms having pseudopodia, generally four in number, sometimes only one being present and then resembling a tadpole in appearance. They were swiftly whirling round and round, often avoiding any obstacle in their way. Bismuth and salol in large doses internally had the effect of stopping the movement altogether and of causing them to assume the spherical form. In some cases the pseudopodia became detached, which began to move and look like filiform snake like bodies. After stopping bismuth, the *amoeba* began their rotation again. The organism has not as yet been described in any books we have come across and we have not met with such organisms any more in stools under the microscope. The whole phenomena were witnessed by Major Calvert, M.D., I.M.S., the then Civil Surgeon of Darbhanga and other medical practitioners of the locality.

CHAPTER VIII.

Symptomatology:—Previous history. General outline. Hepatitis. Fever: Its diagnostic value. Pain:—local and sympathetic; shoulder-pain. Night-sweats. Wasting. Rigidity of the right rectus. Decubitus. Effects on other organs: Lungs—Respiration. Cough. Character of the expectorated liver pus. Pleura. Spleen. Digestive organs—Tongue, Teeth, Stomach, Colon. Peritoneum. Gall-bladder, Jaundice. Vessels:—pulse, retinal hyperæmia. Urinary system. Skin. Nervous system. Local signs:—bulging, enlargement, palpation, percussion.

The patients suffering from abscess of the liver often trace their disease to attacks of hepatitis which often subside and recur for a time before running into formation of abscess. As regards Europeans are concerned, most of the observers agree that history of dysentery (amœbic) is present in the majority. Of the 100 cases, of which we have collected the notes, and who are natives of India, we have got the history of alcoholism 80 per cent. In some there is history of remote dysentery, and in none, have we been able to trace anything like cause and effect. A few suffered from malaria, but the majority enjoyed good health but were

Symptomatology. Previous history.

addicted to drinking. In one case only, there was no history of alcohol, but of *Ganja and Gooly*.

The patient, apparently in good health, after a period of indulgence in alcoholic drinks, suffers from dysentery (in case of Europeans) from which he recovers. But he does not regain his strength. After weeks, he exposes himself to a chill, becomes indisposed, suffers from headache, backache, bad tongue, anorexia, nausea, alternate constipation and diarrhoea and insomnia. He also suffers from irritability of temper and sense of weight and fulness in the right hypochondrium, and hepatitis. Later on he gets an evening rise of temperature with rigors, complains of stabbing pain over the hepatic region and in the right shoulder and of dry cough. His complexion changes to a muddy hue. The fever increases— 102° in the evening, the temperature becoming normal in the morning. He becomes thin, gets night-sweats. He can not lie on the left side; pulse becomes hurried,—80 to 100 per minute. Tongue is foul, furred and yellow. Extremities become swollen, cold and clammy. Breathing shallow and thoracic. The right rectus becomes rigid; the palpation and percussion of the liver become painful. The area of liver dulness increases, its upper line being arched. The attack may lead either to resolution and recovery, or suppuration. A tender spot can be made out

General outline
of a case of
liver abscess.

in the lower intercostal spaces. Spleen is normal. There is pleuritic rub over the base of the right lung, and peritoneal friction over the liver. The urine is scanty, high coloured and deposits urates. In advanced stage, we find an increase of hectic fever, emaciation, sweats, pain and dulness; and after months, he dies worn out, the abscess bursting through lung, pleura, etc.

Hepatitis is often the first stage of the disease. The patient suffers from fever, foul tongue, scanty urine and gastric irregularity. He complains of pain and tenderness over the liver, a sense of weight or tension, increased on pressure. Pain often radiates to the right shoulder. The organ, especially the right lobe becomes uniformly enlarged. It is not a fatal disease. Morehead cured 86 per cent. of his cases. In 1903 there were 354 cases in the English army with 3 deaths only. This condition of the liver is caused by malaria, alcohol and rich diet; and so the organ becomes predisposed to suppuration.

A severe form of the disease, recurs and subsides for a time and then runs into abscess. It may be non-malarious and often occurs in persons suffering from dysentery and diarrhoea but not amebic necessarily. This is rather toxæmia due to the absorption of toxin of amæba.

or bacteria absorbed from the bowels into the liver which has become less resistant or inadequate for the struggle.

Fever may be the initial symptom of liver abscess.

Fever. Sometimes liver abscess appears suddenly

accompanied by severe rigors, high fever and vomiting; and thus simulates bilious remittent fever. Rarely it runs afebrile course; amongst our cases we noticed four patients who did not suffer at all from fever. Pit mentions a case of late abscess which occurred 11 years after his return from the tropics. The fever may be absent and this happened to one of our cases in the S. N. P. Hospital, where a patient was examined by six doctors as to the nature of his enlarged liver. There was no pain, no fever and no history of dysentery. We wrote our diagnosis and we all missed liver abscess. On aspiration, it was found to be a big liver abscess. The patient afterwards admitted the habit of *toddy* drinking. Sometimes after a period of fever, it runs an afebrile course. This is especially seen in chronic cases. Osler¹ says: "One of my patients, with a liver abscess which had perforated the lung, coughed up pus after his temperature has been normal for weeks." In subacute cases again there may be short flashes of temperature at intervals, followed later by steadier fever of a hectic type; and there may be afebrile intervals of several days'

¹ Principles and Practice of Medicine, P. 579.

duration alternating with spells of high temperature. But generally the fever is septicæmic, intermittent or remittent, if it can be said to have any special type. In the septicæmic form, the rigors are severe and irregular (daily twice or once every third or fourth day). The temperature rises to 104° or 105° . In the intermittent type, it is quotidian with chilliness in the morning. More frequently it is remittent with irregular periodical exacerbations: the matinal temperature being 98° , 99° or 100° and evening temperature 101° , 102° , 103° and rarely 104° . Such a range of temperature may last for 2 or 3 weeks when it makes its way outwardly. We saw a case in Dr. Moir's ward, Medical College Hospital¹, where the abscess contained 70 oz. of pus while the temperature ranged between 97° and 99° and in many cases, in which the presence of pus was beyond our doubt, the thermometer showed a normal temperature morning and evening. "Fouquier² observes, that it is a peculiarity of parenchymatous organs, and particularly of the liver, to occasion no fever, even when they are the seat of intense disease".³

When pus is formed, the fever assumes the hectic

¹ Moir. I. M. G. June, 1897.

² Gaz. des Hopetaux, 16th November, 1841.

³ Frerich's Diseases of the Liver, Vol II, p. 127 footnote, Syd. Soc. Trans.

form with constant rise of temperature day after day followed by sweating, just as in pyæmia. This hectic rise of temperature may be long before any actual bulging in any part is noticed. Sometimes this hectic fever resembles ague. We may have again double rise of temperature. In drunkards there is a low remittent fever generally with profuse sweating and furred tongue—this is often the preliminary sign of liver abscess.

In Waring's cases :

Hectic fever in 34 cases.

Cold sweats „ 72 „

Evening rise

of temperature „ 22 „

Prostration „ 74 „

If the chill be decided rigor, the fever high and sweats profuse, it is possibly a pyæmic abscess or large tropical abscess implicating neighbouring organs. The fever of a simple intermittent or remittent type indicates a medium sized abscess, making its way outwardly, with partial injury to the parts traversed. If it be irregular intermittent, with rigors, strong exacerbations and profused sweats, possibly we are dealing with a large accumulation of pus. In chronic cases, the fever assumes the typhoid form. If symptoms of acholia be

Diagnostic
value of fever.

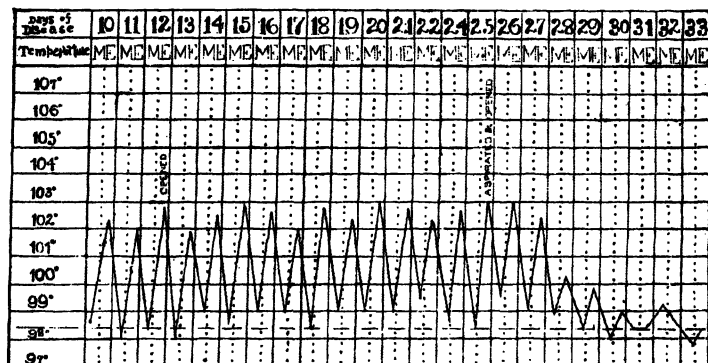
added to the typhoid condition perhaps a large extent of the secreting structure of liver is destroyed.

Classical writers lay particular stress on rigor as an important indication of suppuration. Chevers says:—"There is nothing else in nature like it, so terrible is its severity and so strong the convulsive agitation of the body." Though it occurred in the majority of our cases, it is by no means a constant symptom. Waring finds that rigor was mentioned as present in 25 cases and absent in 22. The fever may come with or without rigors. Generally the evening rise of temperature is preceded by a sense of chilliness or marked rigor simulating the rigor of a quotidian fever. Cantlie thinks that rigor is common in cases which follow upon hepatitis but is rare in post-dysenteric ones.

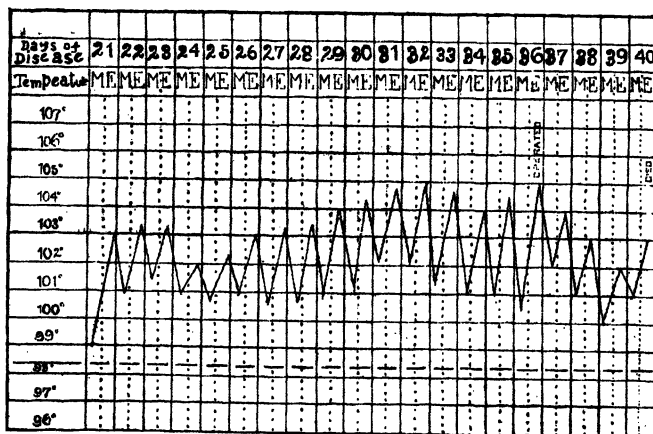
The pain is caused by hepatitis and so may be present in the early stages but may disappear as the abscess develops and may be absent if the abscess be deep-seated. The nearer the pus is to the surface of the liver the greater will be the pain; and so the pain may re-appear when perihepatitis occurs.

Pain may be either local or sympathetic. It is more or less present in some stage. If it is sudden and severe, fever will be present. More frequently a sense

14. Temperature Chart.

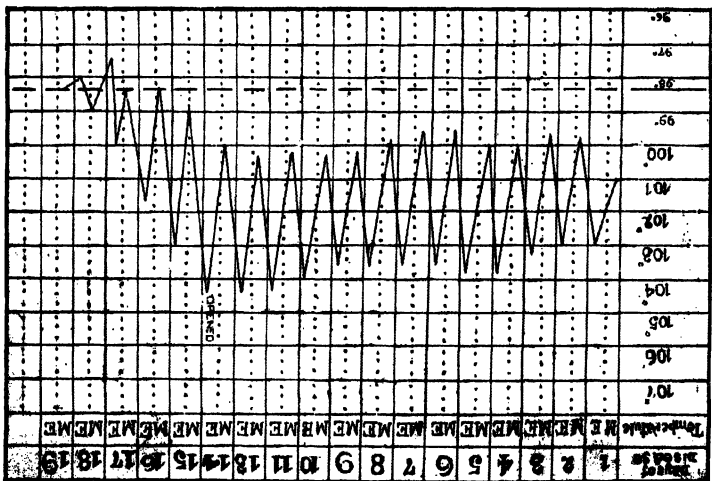


15. Temperature Chart.

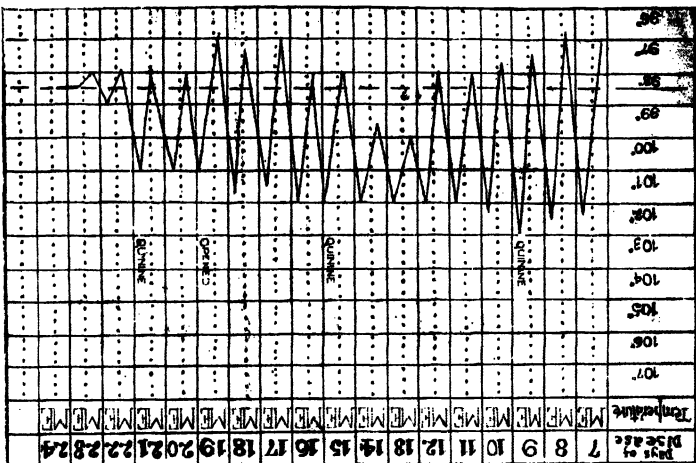


To face page 136

16. Temperature Chart.



17. Temperature Chart



of dragging, weight and tension is complained of, until after suppuration it assumes a throbbing character or the pain subsides. When the capsule is involved, the

Local. pain is of a stabbing kind or stitch-like and is increased by movement as by inspiration, palpation, percussion, succussion or coughing. Chevers¹ says:—
“When there is *inflammation of the hepatic peritoneum*, as there was in my own case, the pain is so great as to render even the slightest movement of the trunk nearly impracticable.” Rouis observed the symptom to be present in 141 out of 171 cases or 85 per cent. The pain on palpation is smarting and shrinking in character, the rectus muscle at once starting up as a rigid protection of the tender parts below. The situation of pain varies it may be in the right hypogastrium, or epigastrium or at the base of the thorax. However pain is not constant and is often absent specially when the abscess is deep-seated in the parenchyma of the gland. “Large central abscess and multiple abscesses do not of necessity cause it” and “it may be taken as a general rule that pain is greatest where the abscess encroaches on a serous surface—pericardium, peritoneum, or pleura.”

¹ A Commentary on the Diseases of India, P. 633.

Shooting pains radiating over the chest and down the right flank and hypochondrium were present in one third of our cases. In abscess of the right lobe, Sullivan found pain in the right shoulder and region of the scapula in one-sixth of his cases. Annesley believed this symptom to be diagnostic of abscess on the convex portion of right lobe. This pain may radiate to the neck or down the arm. If left lobe be the seat, pain in the left shoulder is complained of. The pain may be persistent or intermittent; it may be dull, gnawing, aching, or acute or burning in character. This referred pain is "a reflex transmitted from the hepatic radicles of the phrenic nerve in the neck which receives branches from the fourth cervical to the branches of the cervical and brachial plexus". Waring remarks that this symptom existed in 48 of his 300 cases, and that, in 24 cases, it was noted that it was absent. Budd found this pain in 5 out of 15 cases; and Rouis in 28 out of 163 cases or 17 per cent. Both Louis and Andral deny the occurrence of shoulder pain altogether. Chevers says:—"I saw very little of the right shoulder pain of which so much has been written." In his own case he had no shoulder pain but he continues: "For about two inches above the clavicle, my *right-phrenic nerve* felt like a sensitive cord painfully stretched." Manson mentions a cases who complained of pain on swallowing, at the moment

the bolus of food traverses the lower end of the œsophagus. The shoulder pain merely indicates a perihepatitis—but is not diagnostic of supuration in the liver.

Waring concludes:—"We are entitled to say that pain in the right shoulder is most frequently present when the abscess exists in the right lobe, especially when it is situated in its upper portion, but its presence no more necessarily implies that the abscess is in this lobe, or is in this portion of the lobe, than we are entitled to conclude that this lobe is not the seat of the suppurative process, because pain in the shoulder is absent."

Rheumatic-like pains and swellings of the hands and feet sometimes occur, as in septic affections. They disappear rapidly when the abscess bursts or is opened. The pain in the shoulder may be mistaken for rheumatic pains. Localised tenderness of the liver on deep palpation is a notable sign of the existence and of the position of the abscess. So the whole of the hepatic area should be explored carefully with our fingers.

Nocturnal sweating is often profuse, his clothes being Night-sweats. drenched with perspiration. Even if he sleeps during the day, beads of perspiration appear on the forehead. This may not be marked, or absent; but profuse sweating is the rule especially during sleep. The

sweating is independent of the temperature (Lafleur). The skin remains soft and clammy except during the stage of rigor. Chevers says: "Profuse night-sweating occurred late in my own illness." "Night-sweats are, however, generally among the signs of approaching dissolution in cases of liver abscess."

The wasting is generally decided and progressing ;
 Wasting. the panniculis adiposus becomes scanty, the muscles flacid and bony structures protrude ; this is especially the case when they come for medical help in the hospital. A state of exhaustion is gradually developed in most cases but in private patients the flesh is often well retained. Rouis records a few cases in whom a great increase of fatty tissue was observed to take place.

In some cases paralysis and wasting of the deltoid and other arm muscles, has followed an abscess of the liver, but the exact connection between them is not clear.

The right rectus muscle remains in a rigid
 Rigidity of the condition which becomes marked during
 right rectus. percussion or palpation. This symptom was noticed by Twining and was considered as a pathognomonic sign of deep-seated abscess of the liver. It is purely a sympathetic affection, the result of an impulse to protect the inflamed organ, and is met with in other diseases besides abscess of the liver, as in inflamed gall-bladder,

Waring only finds it noticed as being present in 5 out his 300 cases, and Chevers mentions it as a rare occurrence.

He can not lie on either side for pain. Generally he Decubitus. prefers to lie on his back, with the shoulders raised and the knees flexed. The decubitus is usually dorsal or right dorsal, the body being bent somewhat towards the right and the right leg is drawn up. The gait is also peculiar; on standing, he stoops to the right and forward due to an "involuntary protective impulse." He can not lie on his left side, as it causes pain from the dragging on the adhesions formed between the liver and adjacent structures, or from the discomfort caused by the pressure of the enlarged liver on the heart and stomach; therefore he prefers to lie on the right side. But the decubitus is variable; and sometimes patients lie on their left sides, especially in acute cases before the formation of adhesions. Most of the cases observed by us used to lie on the left side in acute stage possibly to avoid the liver being pressed, but towards the later stages, they changed their positions, lying more on the back than on the right side. Chevers says: "I only found comparative ease when lying on my back".

In Waring's cases = 35.

10 could not lie on the left side.

4 " " " " " "

3	could	not	lie	on	the	right	side.
3	„		„	„	„	„	„
3	„		„	„	„	either	„
2	„		„	„	„	„	„
2	„		„	„	„	back & left	„
1	„		„	„		at all.	

The formation of tropical liver abscess produces marked changes in organs which enter into relations with liver, and so the various effects are to be considered.

In the lungs, we find symptoms of basal pneumonia
Lungs. before rupture of the abscess into the lung.

The congestion is due to the arrest in thoracic movements over the painful area. The abdominal breathing gives place to thoracic, and the lower ribs remain immobile, consequently blood stagnates in the basal part of right lung; and inflammatory changes in the lower part favour consolidation. It is of sub-acute type and persistent in character, with signs of consolidation and crepitant rales on inspiration. These signs indicate contiguity of the abscess to the diaphragm. After rupture, the sputum is characteristic, and on examination of the chest, we detect bronchophony, bronchial breathing, defective resonance, feeble tubular breathing and increased tactile fremitus.

Respiration is shallow, thoracic, short, rapid and

catching. It is short and catching when diaphragm is involved and painful if pleura is inflamed. The breathing is short and rapid due to the attendant fever and also because fuller inspiration is attended with a stitch. Breathing on the right side is entirely thoracic and the diaphragm is almost motionless.

Cough is a constant symptom and is short, dry and hacking in character. "*Tussis arida, sicca, molesta quidem, sed rara*" (*Hippocrates*). It is "due to a reflex from irritation of the diaphragmatic pleura or from an inflamed condition of lung or pleura over the abscess, or to oppression or dyspnoea from the bulging upwards of diaphragm." Afterwards when the abscess discharges through the lungs, cough is catching, severe and painful, and may be with vomiting or purulent expectoration. Another symptom, singultus or hiccup belongs to the period of discharge and is often extremely protracted and exhausting. Louis thinks that cough when present is due to bronchitis, but we could not detect any signs of bronchitis in many, although they suffered severely from cough; thus proving the sympathetic nature of the symptom.

The respiration may remain quite normal in some cases of hepatitis, especially when the pus is deep-seated and when neither the diaphragm nor pleura is implicated in the disease.

The character of the expectorated liver-pus is pathog-
 Character of the
 expectorated
 liver-pus. monic of the disease. Suddenly the patient
 expectorates brick-red puriform matter,
 containing debris of liver tissue, sometimes tinged
 yellow from bile, with the physical symptoms of basal
 pneumonia. The brownish tint of the sputum is due to
 blood pigment and altered blood corpuscles; and
 there may be orange-red crystals of hæmatoidin. After
 a few days, it becomes brown, viscid and jelly-like, and
 streaked with red blood. The blood comes from the
 abscess wall due to injury caused by the harassing cough.
 The pus sometimes contains *amæba colli* (Ruse and
 Laffleur). In one case, we found *amæba colli* in the
 expectorated liver pus, identical with those which exist
 in the liver pus and in the stools. They were in number
 and displayed active amæboid movements under the
 microscope.

The pleura is sometimes inflamed by infection through
 Pleura. the diaphragm before rupture. Friction
 sounds and symptoms and signs of pleurisy indicate this
 complication. The friction sounds due to perihepatitis may
 thus become obscured.

Enlargement of spleen is as a rule a rare complication.
 Spleen. of tropical abscess for the portal circulation
 is not interfered with by the disease. The spleen may be

found to be always small. We found however enlarged spleen in five of our cases, but they suffered from previous malarial infection.

Tongue: The tongue may remain clean and a smooth red tongue with fissures is not uncommon ; but it is usually coated and furred at the onset, but in protracted cases it may become clean or is only slightly foul. In some few cases it is brown, dry and glazed in parts, whilst covered with a grey or yellowish membrane-like crust at the base and margins, a condition often present at the onset of suppuration. Haspel and other French physicians have not confirmed the opinion of Annesley who thought that the projection of red papillæ through a grey coating of the tongue, which ultimately become brown or brick-red, was a diagnostic sign of inflammation in the liver. Neither was any critical salivation observed to accompany hepatitis, as maintained by Portal and others.

Teeth: Aphthæ and sordes on the teeth are a late sign and usually foretell a fatal termination.

Stomach: Loss of appetite is marked from the beginning. The patient often vomits either due to pressure of the enlarged liver on the stomach or as an expression of gastric catarrh. Obstinate vomiting a symptom of ominous import in the prognosis is present in many cases

and ought to excite suspicion in a tropical climate. If the walls of the stomach or abdomen be affected, vomiting may recur until pus escapes into other channels. Vomiting or nausea was present in 74 of Waring's cases, and absent in 10. Fayrer and Maclean attach diagnostic importance to the frequency and persistence of the vomiting. Bartholow confirms them: of 84 cases he found this symptom in 74. In our patients, however, vomiting was not so marked a symptom—it was present in 27 per cent. of the cases.

Vomiting is more frequent when the abscess is in the left lobe or when it occurs in both lobes. Rarely vomiting may be due to the abscess pressing on the pylorus and leading to pyloric obstruction. Flatulence and eructation are commonly present.

Colon: Bowels: The patient often complains of flatulence which may be troublesome. Bowels are either confined or irregular; or there may be diarrhoea and dysentery due to ulceration of the intestine caused by septic absorption from the abscess. A liver abscess frequently finds its way in the colon; and there distension of cæcum and ascending colon from gas may occur just before perforation. The bowel may then be constipated or may be the seat of an imitating flux. Of 143 cases observed by Rouis, in 30 only or 21 per cent. there were present symptoms of gastro-enteric catarrh, anorexia

nausea, vomiting, etc. Sometimes hepatic and dysenteric symptoms, if concurrent, alternate in severity. If amæba be found in the stool, it is a valuable diagnostic sign. In our cases we neither found amæba, nor dysenteric symptoms running concurrently with the hepatic lesion, except in one. Dysentery often supervened after operation of the abscess and it was often the fatal symptom—the precursor of death.

Stools : Clay-coloured stools are rare except in large abscesses implicating the liver tissue. In only one of Waring's 300 cases, the stools became white and deficient in bile.

Diaphragm : Risus Sardonius : Chevers detected its very marked presence in one of his "European patients in whom hepatic abscess progressing upwards had eventually caused an unusual amount of diaphragmitis." Hiccup is a frequent symptom.

In 2 of Waring's cases, a liver abscess, had carried the diaphragm up to the level of the 4th rib ; in other 2 cases, it reached the 3rd rib, and in one, it reached the level of the 2nd rib.

Peritoneum : Sometimes peritoneal friction is to be made out. Peritoneal and pleuritic friction sounds, if audible, sometimes may be availed of as localising signs.

Gall-bladder :—Jaundice is a rare symptom of tropical Jaundice. abscess of the liver as gall-bladder is seldom encroached upon. “Jaundice is very seldom present in [Indian] hepatitis.” (Morehead). “It did not occur in any of my own Bengal cases.” (Chevers).

Observers.	Jaundice	cases.	Per cent.
Cassimir	} 23	66	34
Broussais			
Rouis	26	155	17
Thierfelder			16
Waring	2		6
Morehead	5	120	4

But a hepatic abscess may press upon the main bile duct downward and so cause jaundice.

Ascites, oedema of the lower extremities, enlargement of the superficial veins of the abdomen and hæmorrhoids are rarely noticed in tropical abscess of the liver. This should be remembered, for a large abscess may fill the greater part of the abdomen and has given rise to erroneous diagnosis of ascites. Haspel noticed ascites in two cases, produced by closure of portal vessels due to pressure. Varicosity of the epigastric and hæmorrhoidal veins—one or both of them—is sometimes observed.

The pulse of the patient is neither hard nor very quick, but generally has an irritable throb, indicative of some internal malady. But in the cases with high fever, it may be hundred and forty or more. In our cases, the number of pulse was often 120 to 140 per minute. Exceptionally the pulse may be slowed.

In 15 cases in whom we examined the eyes with ophthalmoscope we were struck with the constant presence of retinal hæmorrhage which caused discomfort to the patients in the use of the eyes—a complaint often made by patients suffering from liver abscess. The ophthalmoscope frequently shows premature senile degeneration of the lens, manifested by striæ occurring in the extreme periphery. Junge,¹ Stricker, Buchwald-Forester and Litten have all recorded cases of retinal hæmorrhage in cases of grave disease of the liver; and surely the value of this sign requires investigation. There is also a decided contraction of the field of vision in some cases.

The urine becomes scanty and high coloured or tinged with bile and “almost invariably produces considerable scalding in its passage through the urethra” (Martin). Dysuria is noted in 8 of Waring’s cases. It often deposits a pink sediment and often

¹ Heinrich Muller’s Gesammelte Schriften. P. 331-335.

becomes turbid a few hours after it is voided. There may be excess of urates and uric acid but it is deficient in urea. But when the fever is high, the urea may be in excess. It contains also leucin and tyrosin. Albumen is not commonly present. From absorption of pus, albumen may be found. Albumenuria occurs sometimes. The specific gravity is high. Indican may occur. These pathological changes in the urine are not at all characteristic of liver abscess, except where it contains pus.

In a case following dysentery, crystals of leucin, tyrosin and cholesterin were present in the urine for 10 weeks, and disappeared after opening the abscess. Pfähler found tyrosin-like crystals of a soap of lime and magnesia in one case.

Semmola and Gioffredi say: "Important diagnostic signs may be found by a daily examination of the urine. In the first stage of the disease there is a certain increase of urea, which is dependent upon inflammatory irritation in the hepatic cells. In the second period, that is to say, when the fusion of purulent foci has occurred from destruction of a considerable portion of the parenchyma, we have hypoazoturia. This is a symptom which has always been of especial interest to us, and which we can affirm to be constant, as we have found it in every case of hepatic abscess in our practice, and regard it as a valuable aid to

diagnosis. It is of the more importance because the elevation of temperature in this affection, should produce and increased amount of urea, for which reason the inverted relation between the thermic condition and the urea must, in connection with other symptoms of liver trouble, be of great practical value. In the calculation of the daily amount of urea the quantity of urine eliminated in the twenty-four hours should always be exactly estimated, for as the urine is usually reduced in amount, this symptom might not otherwise be appreciated at its full value."

The skin has not the natural moist, soft feel of health ;
Skin. it is often dry, with clammy perspiration, irregular flushes and chill. The temperature of the surface is never much increased.

The complexion is generally muddy, cachectic and
Complexion. slightly icteric-looking. In some cases, the skin is sallow or dusky, and the eyes, yellowish ; but this may arise from blood disintegration. This may be true of Europeans, but the natives rarely show any tinge of jaundice. The face is pale, the conjunctivæ are infiltrated and often slightly bile tinged. There is in faces and general appearance, a strong suggestion of the liver abscess. The anxious countenance is seldom absent. "A glance at one of these pinched anxious visages affords

us a lasting memory which can always be applied again" (Chevers).

The nervous system. Sleep: Disturbed sleep and Sleep. frightful dreams are often the accompaniments of the disease and are present in almost every case.

Mind loses its firmness. The patient becomes gloomy and irritable. He suffers from excessive Mind. and unaccountable languor and depression of spirits. Hypochondria is present in many cases (Hammond). He justifies aspiration from this symptom alone. There may also be stupor, hebetude of mind, confusion due to acholia and cholæmia. The state of the mind may be referred to the action of the poison, absorbed from liver abscess on the cerebral cortex. Rarely pyæmic abscess in the brain or septic meningitis occurs. Delirium or rarely convulsions may occur before death.

Local œdema, in advanced cases, over one or more Local signs. intercostal spaces or extending over the hepatic area is detected. When limited, it is a very useful localising symptom. But redness of skin is observed only when the abscess approaches the surface. Œdema over the abscess was only present in about 5 per cent. of Stevenson's cases.

Local bulging occurs usually in the epigastrium, or Bulging. right hypogastrium but pus may burrow

down the flank or among the muscles of the abdominal wall. Again bulging may be absent though there may be enough pus formation ; the bulging may be upward, scarcely ever downward. The abdomen is fully distended, as if from tympanitis, and a rounded tumour may be detected at the site of the liver.

Fluctuation is only to be made out when the abscess is near the surface ; If fluctuation occurs with bulging, we are more sure that pus is superficial and the abscess is pointing. "Another distinctive character of abscess is that the fluctuation is usually surrounded by a mass of inflammatory hardness" (Murchison). But a large deep-seated abscess may cause no apparent bulging, fluctuation or even enlargement. Bartholow could detect fluctuation in three out of twelve cases. In ten cases, we were sure of it in 4 only in advanced stage. Malbut ¹ considers painful succussion a sure sign, but we did not find it in the majority of our patients.

Enlargement of the liver is a distinctive sign but is often late in appearance. In Waring's cases there was evident enlargement in 90, and no enlargement in 11. Rouis noted enlargement of the organ 73 times in 122 cases or in 60 per cent. but taking the results of the post mortem examinations the liver was enlarged in 70 out of 101 cases or 70 per cent.

Now the question may be asked: "At what time does the enlargement occur"?

Rouis in 122 cases—mostly at the onset of suppuration.

Louis in 51 cases

- 12 ... before suppuration.
- 22 ... at onset of suppuration.
- 17 ... after suppuration.

Bartholow in 49 cases.

- 2 ... before any symptom.
- 8 ... at onset of symptoms.
- 39 ... after other symptoms.

Palpation: The patient complains of pain—the hepatic pain, and we may appreciate perihepatic friction. We can feel fremitus on deep inspiration and the extent of enlargement of the organ. The enlargement of the liver is most marked in the right lobe as its upper surface is the usual seat of abscess. The increase in volume is upwards and to the right and the dullness may in extreme cases reach as high as the 2nd rib; it is not downwards, as in cancer, cirrhosis, lardaceous disease, and other affections producing enlargement of the organ. But sometimes the enlargement causes bulging of the right side and the edge may project a hand's breadth below the costal border. Then we find the surface of the liver which is

usually smooth to be indurated and elastic and sometimes fluctuation can be easily detected there, though to tell the truth, this symptom is rarely found. In such cases, the underlying colon sometimes masks the hepatic dulness. When the abscess is situated in the middle line of the body, it may pulsate from transmission of impulse from the aorta. Carpenter describes such a case.

Inspection: On inspection, we can detect an enlargement of the right hypochondrium or a protrusion of the ribs, or a circumscribed bulging of the right costal wall with widening of the intercostal spaces, or a swelling on the right costal arch, or in the epigastrium. If the abscess bulges to any extent, it may be discernible as a distinct prominence in the epigastrium or right hypochondrium. A more pronounced curve of the lower-ribs, and protrusion of the lower intercostal spaces can be easily detected. In some cases the inferior border of the organ projects and its respiratory movements may be seen. Rarely the thoracic wall looks cedematous and the sub-cutaneous veins become prominent.

Percussion: a common sign of enlargement of the liver is the upward extension of liver dulness, "so much so that Sachs consider this to be a diagnostic sign." The upper boundary of the abscess becomes convex or domelike after it has made some progress. But

extension may be upward, downward or horizontally. The line of dulness is often characteristic—it is normal at the mid-sternal and para-sternal lines, begins to rise at the nipple line, reaches the 5th rib at the mid-axillary line, curves behind the angle of scapula and on approaching the spine, markedly tends downwards. But this boundary of the dulness would not be true if the seat of abscess be in the left lobe. But the abscess may be so small and so deeply seated that percussion will not elicit any extension of dulness beyond the normal limits. Percussion may convey to the examiner a sensation of ballotment, as in the case of a thick-walled elastic bag filled with air.

Auscultation: We detect friction sounds of pleurisy or Auscultation. crepitations of pneumonia. The friction sound is due to perihepatitis, or may be sometimes felt by the hand. Fine crepitations occur with inspiration and expiration. Bertrand regarded it as caused by perihepatitis but Hasler and Boison have heard it in cases where laparotomy showed absence of perihepatitis and think it is produced in the softened and œdematous liver substance around the abscess. When the abscess approaches the abdominal wall, the peritoneal friction sounds become audible over the apex of the tumour at first; gradually the area of friction shall widen as the abscess increases in size, but finally the friction sounds may disappear in the central parts due to

adhesion having taken place or to an effusion of lymph, between the peritoneal surfaces. Cantlie lays stress on this observation of the central non-frictional area as the safe site of operative interference.

Measurement: A difference in circumference of half inch to one inch may be detected even when the enlargement is not marked.

Smell: Hatch smelt a strong hepatic odour from a patient, though we never detected it.

CHAPTER IX.

Duration of Liver abscess. Its terminations; absorption, rupture into lungs, pleura, stomach, intestines, pericardium, peritoneum, blood vessels, right kidney, and skin. Complications. Mortality, cause of death. Diagnosis, Prognosis, Recurrence.

The duration of liver abscess varies within wide limits.

We shall see from the table below that we
The duration of liver abscess. can not fix any probable duration of liver abscess. Rose and Carles say: "Occasionally it remains passive as a chronic encysted abscess, and then the walls become very thick. One of us recently opened an hepatic abscess which had been diagnosed by a hypodermic needle twelve years previous and left alone. It contained about 2 pints of pus and the walls were fully half inch thick. The patient came under observation because the swelling was more prominent as the result of increased intra-abdominal pressure, due to pregnancy." But such cases are no doubt exceptional. The mean duration of our cases is 90 days if left without surgical interference.

Rouis, after analysing his extensive materials has arrived at the following results¹ :—

¹ *Frerichs: Diseases of Liver*, vol. II, p. 139. Syd. Soc. Trans.

"The cases which terminated fatally, lasted on an average :—

A. When the abscesses did not open
externally 70 days.

B. When the abscesses discharged their contents
externally :—

1. Through the abdominal or thoracic
wall 70 days.
2. Directly through the bronchi ... 125 „
3. Through the bronchi after
previous discharge into the
pleura 185 „
4. Through the stomach ... 150 „
5. Through the colon and bile-
ducts Some months.

The average duration of the cases included under the
series B, was 110 days.

The duration of cases which recovered was as follows :—

1. Where the abscess burst
through the thoracic, or
abdominal wall 140 days.
2. Where the abscess burst through
the bronchi 115 „
3. Where the abscess burst through
colon 140 „
4. Where the abscess burst through
stomach 180 „

The average duration was 140 days. . .

Observer.	Cases.	Nature.	Period.	Average duration.
Waring	220	39 days. (59 from 10 to 20 days).
Roux	179	...	Shortest 10 days Longest 480 "	60 days (104 from 11 to 60 days).
Murchison	"	...	2 month to a year	...
Davidson	Usually several weeks or months, may be chronic. Rarely fatal within a week or two.	...
Manson	Many 3 weeks, often several months; may be a year or longer if it bursts through lung	3 weeks.
Fayer	"	3 weeks, from the onset of symptoms.
Bartholow	"	...	2 weeks to 6 months.	...
Oster	6 to 8 weeks to several years.	...
	10	Dysenteric	...	19 weeks from the day of hepatitis
	16	Idiopathic	...	6 weeks.
Kelsch Keiser }		...	Died 6 days after admission and he had been suffering from a few days only.	...

The tendency of an abdominal abscess to external Termination. discharge has been observed from very early times. Caraka and Suśruta mention two ways by which such an abscess can discharge itself namely by the mouth and per rectum. The abscesses of the upper part tend to discharge through the lung, those of the lower part through the natural opening below. Abscess of the liver comes within the former rule, and the modern experience shows that it is one of the favourable modes of termination. The course of the pus is determined by the course of the lymphatics, and as the majority of the lymphatics of the liver find exit upwards between the layers of the coronary ligament, the path of the pus is in the majority upwards. As regards the various modes of termination of liver abscess, we have the following statistics to guide us. Rendu's statistics show 159 cases of spontaneous rupture out of 563 cases *i.e.* 20 per cent.

Rendu:

162 cases fatal.

96	Pus confined in the liver.
66	{	16	...
		50	...

Multiple abscess of which a portion had burst.

Pus escaped outside the liver.

162

Pus came in contact with :—
 Diaphragm.
 Pericardium.
 Stomach.
 Pancreas.
 Gall bladder.
 Colon.

26	...	Pus escaped into the cavities of
14	...	Peritoneum.
11	...	Right pleural cavity.
1	...	Pericardium.

[In one case pus escaped both into the pleural cavity and peritoneum. In another case, it also escaped externally.]

30	...	Pus escaped externally.	
15	through lungs and bronchi	...	50 per cent.
2	... abdominal wall	...	6.6 " "
2	... pleura and chest wall	...	6.6 " "
5	... stomach	...	16.6 " "
4	... intestines	$\left\{ \begin{array}{l} 1 \text{ into duodenum} \\ 3 \text{ ,, transverse} \\ \text{colon} \end{array} \right\}$	13.3 " "
1	... bile duct	}	6.6 " "
1	... gall bladder		

[One contained 2 abscesses, one opened into colon, the other into stomach.]

7	...	Opened artificially.
<hr/>		
69		

39 cases of recovery :—

17	burst through abdominal or thoracic wall.
$\left\{ \begin{array}{l} 3 \\ 13 \\ 1 \end{array} \right.$... right last intercostal space.
	... epigastrium.
	... umbilicus.
15	... bronchi.
3	... stomach.
4	... transverse colon.
<hr/>	
39	

Theirfelder — 170 cases.

Lung	...	74	} 59 per cent.
Pleura	...	26	
Pericardium	...	4	
Peritoneum	...	23	
Stomach	}	13	
Duodenum			
Intestine	...	32	
Kidney	...	1	

Cyr:	159 cases.	per cent.
Lungs:	59	" 10·5
Pleura:	31	" 5·5
Pericardium	1	" 0·13
Peritoneum	39	" 6·9
Stomach:	}	8 " 1·4
Duodenum		
Intestine:	13	" 2·3
Kidney:	2	" 0·3
Inf. vena cava	3	" 0·5
Bile passage	4	" 0·7
Externally	2	" 0·3

162.

Perforation may take place in more than one site in the same case.

Bartholów:	12 cases.	per cent.
Lungs:	5	" 41·6
Stomach	}	4 " 33·3
Intestine		
Externally	3	" 25

12

Haspal:	25 cases opened.
7	externally through the abdominal or thoracic wall.
2	... lung.
4	... pleural cavity.
2	... peritoneum, in one case pus escaped into the scrotum.
10	... pus confined in the liver.

25

Cambay:	10 cases:
1	" opened into the peritoneum.
1	" ... gall bladder.
2	" ... bronchi.
6	" ... Pus confined in the liver; in one was commencing cicatrisation.

10

Morehead:	140 cases:—
14	opened into the lungs or pleura. 10 per cent.
	... stomach, intestines 3·5 "

[Recovery—3.]

2	...	peritoneum.
21	...	no rupture but circumscribed
7	had	peritonitis.
14	"	secondary general peritonitis.
5	...	pus was absorbed.

Andral: 11 cases:—

9	"	...	pus confined in the liver.
1	"	...	opened into stomach.
1	"	...	" peritoneum.
<hr/>			
11			

Waring :	300 Cases.	per cent.
Remained intact ...	169	56·335
Evacuated by operation, a solitary abscess being present ...	129	} 16·000
Evacuated by operation, there being numerous abscesses, one opened and the others remaining intact ...	18	
One abscess opened by operation, another subsequently bursting into abdominal cavity ...	1	
Opened spontaneously into the thoracic cavity ...	14	4·666
Opened spontaneously into the right lung ...	28	9·333
Opened spontaneously into the abdominal cavity ...	15	5·000
Opened spontaneously into the colon or large intestine ...	7	2·333

Opened spontaneously into the stomach	1	} 6333
Opened spontaneously into the hepatic vein leading to vena cava	2	
Opened spontaneously into the hepatic vein at its junction with the vena cava and another communicating with cellular tissue around the right kidney ...	1	
Communicated with the hepatic duct	1	
Communicated with the right kidney	2	
Communicated with the gall-bladder	1	
Communicated with the abscess in the iliac region	1	
Opened spontaneously through the rib in the back	1	
One abscess had opened into colon, another had passed off by the hepatic duct into duodenum ...	1	
One abscess had opened into stomach, a second into duodenum, and a third had been evacuated by operation	1	

One abscess had opened into abdominal cavity, a second into lung	1	} 6·333
Terminated in erysipelas of the lower extremities stimulating phlegmasia dolens, and abscess opening into lungs	1	
Doubtful	5	
	300	100·000

Thus we have only 83 cases in Waring's list for purpose of comparison, as they opened spontaneously.

Thoracic cavity	42	
Right lung	28	
Abdominal cavity	15	} 23
Stomach	1	
Intestine	7	
Externally	2	
Other ways	16	

The liver abscess terminates in various ways.

Below, we have considered the different methods of terminations described.

1. *Absorption*: It is probable that the pus from small abscesses may be absorbed, and in some cases contracted cavities with cretaceous remains have been found in the liver in post mortem examinations, indicating a former abscess.

Cases are recorded in whom liver abscess became encysted, its contents undergoing more or less complete absorption. In the pathological collection of the Calcutta Medical College, the preparations No. 532—34 illustrate the formation of cysts in the liver tissue. Chevers, and Mackinnon report such cases. The encysted abscess may be irritated and so form another abscess. Macnamara says: "These cysts, with their contents, presented the happiest termination of liver abscess, but they appeared to have become the focus of a new congestion under the disturbing influence of the attack of dysentery of which the patient died." "Rouis doubts the possibility of such an event whilst Haspel Catteloup, Cambay (*Dysenterie de la Province d'Oran*, P. 323, Obs. 34 and 37), and particularly Morehead "record observation, which, in their opinion, demonstrate the possibility of this mode of cure" (Frerichs).

2. *Rupture into the lungs*: It is preceded by the symptoms of inflammation at the base of the right lung—dulness, bronchial breathing, rusty sputum, pain, &c. Then the patient suddenly coughs out, mouthful after mouthful, thick pus with blood, and shreds of hepatic tissue. Rouis collected 900 grammes or 20 ounces of bile expectorated by a patient during 24 hours. Sometimes sudden death occurs by the flooding of the lungs with pus—the first gush may suffocate the patient. Commonly, discharge occurs

gradually, the quantity of pus being 5 to 10 oz. in 24 hours. The breath often acquires foetid odour. If the cavity in the lungs is superficial and empty, cavernous breathing may be audible. In favourable cases, the daily amount diminishes, and recovery is possible. Frequently, after a delusive lull, expectoration returns with other signs of septic absorption. This process may continue for a time until the patient is worn out. Sudden death may occur by hæmoptysis due to ulceration of large pulmonary vessels. The lung may also be the seat of an abscess. In some cases, the expectoration never ceases; and if fever be present, we may be sure of a second abscess. Bartholow says that "sometimes before the abscess really reaches the diaphragm, preparation is made in the lung for the discharge through a bronchus".¹ He cites a case mentioned by Dickinson,¹ in which, an abscess holding about four ounces of pus was contained in the upper part of the right lobe; its walls were irregular and not lined by a limiting membrane. It is further stated that the right pleura was coated with flocculent lymph, and the cavity contained serous fluid. Here in advance of the abscess, preparation was made for its discharge through the lung. This grave accident occurred in 14 of Waring's 300 cases.

3. *Rupture into Pleura*:—We get signs of pleurisy,

¹. Pepper's System of Medicine vol. 11. P. 1014.

with a dull pain and slight dyspnœa, which unless relieved by drainage, the discharge may give rise to empyæma or may rupture externally through the lung or the wall of the chest. The patient complains of acute pain and dyspnœa. In the former case the pus passes into the bronchial tubes (Waring has collected 10 of these cases which ended in recovery), and in the latter case a flat tumour is formed in one of the intercostal spaces, which opens externally afterwards.

4. *Rupture into the Stomach*:—The patient complains of gastric pain or irritation, and vomits the characteristic pus. The pus may pass out by the bowel. There would be a temporary cessation of local symptoms and fever. This complication appears to be not very rare.

5. *Rupture into the Intestines*:—This accident occurred in 8 out of 25 cases of spontaneous recovery collected by Waring. Coincident with the sudden subsidence of the tumour, the patient gets diarrhœa passing altered pus in his stool. Two cases are related by Dufresne¹, in which, a painful swelling of the liver subsided after evacuations of blood and pus per anum. If the pus escapes into the bile ducts or the duodenum, it is difficult

¹ Trans. of the Path. Soc. vol xxxii, p. 127

² Durchbruch von Blut and Eiterherden der Leber nach dem Darmkanal, Schmidt's jahrb. vol. 101. P. 51.

to recognise pus in the stool. Cases of communication with the duodenum are noted by Waring and Leith.¹

6. *Rupture into the pericardium* :—This is by no means a very rare mode of termination. None however occurred in Waring's 300 cases though he gives references to six cases in whom this most disastrous complication happened. It occurred only once in Rendu's statistics. Carter reports two cases.² This termination is always fatal. Usually death is instantaneous, except in cases where the opening is at first minute. The patient complains of violent pain over the cardiac region and a sense of suffocation. There would be other signs of pericardial effusion ;—increased præcordial dulness, friction sounds with tumultuous action of the heart and dyspnœa. The accident is especially liable to occur where the abscess is in the left lobe. Tolot³ reports a case where abscess in the left lobe set up sub-phrenic abscess which ruptured into the pericardium. Rokitansky, Graves and Fowler report cases. Cuaran⁴, Nolan⁵, Dymock⁶ and Ewart⁷ mention similar occurrence. Rolleston has notes

¹ Leith's Deaths in Bombay 1855.

² Bombay Med. and Phys. Trans. 1853, p. 305 ; No. 8. N. S.

³ Tolot. Lyon Medical. 1902, p. 51.

⁴ I. M. G. vol. XV, Nos. 9 and 12, and vol. XVI, Nos. 2 to 4.

⁵ I. M. G. vol. II, p. 177.

⁶ I. M. G. vol. I. p. 32.

⁷ I. A. No. XXI, P. 308.

of 9 cases. Bentley¹ mentions a case in which 4 pints of sero-purulent fluid was in the pericardium.

7. *Rupture into the peritoneum*:—This may occur freely into the general peritoneal cavity or may give rise to a localised intraperitoneal abscess which may burst again into the general cavity. Perforation into the general cavity is often the result of traumatism or sudden exertion. It is nearly always fatal. Halke² has recorded a case of recovery. We detect signs of acute peritonitis with sudden accession of pain. It occurred in 14 out of 162 cases (Rouis) and in 11 per cent. of Waring's cases. The patients die within a few days; sometimes circumscribed collections of pus are formed in the peritoneal cavity, which may open externally at the epigastrium (Rouis) or between the 11th and 12th ribs (Cambay) or through the inguinal canal (Haspel). Peritonitis may occur, independently of any rupture, by the propagation of the inflammatory process from the liver. Similarly pleurisy and pericarditis may occur (Morehead). The average duration of life after this accident is about 3 days according to Waring. In one of Twining's cases the patient survived 26 days. It is inevitably fatal.

8. *Rupture into the blood vessels*:—This can seldom be recognised during life.

¹ Bentley. Trans. Path. Soc., vol. II. p. 70.

² Halke. Medico-Chirurg. Trans vol. lxxvi. p. 81.

(a) *Rupture into the portal vein*:—It is extremely rare. Sometimes thrombosis occurs in the vein when the abscess is in close contact with it, and thus rupture into the vein is prevented (Rolleston).

(b) *Rupture into hepatic veins and inferior vena cava*—The hepatic veins have no fibrous sheath, so leakage into them is more common than in the branches of portal veins which are surrounded by Glisson's capsules. Rarely the abscess has perforated directly into the inferior vena cava and not into the hepatic veins. There are 3 cases in Cyr's list. Rolleston mentions and figures a case in St. George Hospital in which and in other cases also a parietal thrombosis was set up and for a time prevented the entrance of pus into the circulation. The pus eventually passes into the circulation and gives rise to secondary abscesses in the lung or to pulmonary embolism.

Flexner¹ has reported two cases of perforation into the inferior vena cava. Perforation of the ascending vena cava or of the hepatic vein occurred in 2 per cent. of Waring's cases. In one of Webb's cases a liver abscess opened into the hepatic vein at its junction with the vena cava.

9. *Rupture into the pelvis of the right kidney*:—The patient discharges pus in the urine by the urethra. This

¹. Am. Jour. of Med. Sc. vol. cxiii, p. 553, May, 1897.

event, mentioned by Annesley, is however very rare. Webb¹ reports a case where a liver abscess made its way through the structure of the kidney into the lumbar region. In Waring's collection, two cases are said to have communication with the right kidney. He numbers the recorded cases as ten².

10. *Rupture through the skin*:—This is said to be the most favourable, though a rare termination of liver abscess; the pus may burrow under the skin for some distance and point into the axilla, as recorded by Portal³ or making its way along the suspensory ligament, emerge at the navel or descending, appear in the lumbar region or under Poupart's ligament, but the most common site is at the epigastrium, or in the right hypochondrium. Many abscesses point between the ribs at their greatest bulge. Gioffredi mentions a case in whom a perforation occurred through the pleuritic adhesions in the infrascapular region in the 8th intercostal space.

11. *Rupture into the gall-bladder*: This accident occurred in one of Waring's cases. It is rare. The pus may eventually pass down the common bile duct into the duodenum and so imitate rupture into bowels. Symptoms of biliary colic have been noted in such cases.

¹ Ewarts' Catalogue. P. 123.

² Diseases of Liver. P. 146.

³ Anat. Med. t. v., P. 304.

12. *Evacuation of liver abscess through a bile duct:*

This complication has been repeatedly observed.

13. Abscess may remain intact and yet the patient may die. In Waring's 300 cases—169 remained intact. In 203 fatal cases of Rouis, 96 were within the liver. Theirfelder noted it in half the number of his cases.

“Of 203 cases collected by Rouis 162 terminated fatally; in 39 there was a complete, and in 2, an imperfect cure; thus the cures were 20 per cent., and the deaths 80 per cent”. The fatal termination was brought about in various ways, described hereafter.

We have some very excellent pathological preparation of liver abscess showing its terminations in the Museum of the Medical College, Calcutta.

Series IX. Part V.

- 278. Cicatrix of an old abscess of the liver.
- 286. Abscess of the left lobe.
- 287. Enormous encysted abscess of the right lobe.
- 288. Abscess of the right lobe—pierces diaphragm and opens into the pleural cavity.
- 289. Abscess of the right lobe—pierces diaphragm and opens into base of the right lung.
- 290. Abscess of right lobe—pierces diaphragm and opens into the transverse arch of colon.
- 291. Encysted abscess.
- 292. Liver and right lung adherent to diaphragm—the abscess communicating with bronchial tube.
- 293. Abscess of left lobe—rupturing through diaphragm into pericardium.
- 294. Abscess of the right lobe.
- 295. Rupture of an hepatic abscess into the stomach.

- 296. Abscess of the right lobe--perforating the diaphragm and involving the base of right lung.
- 300. Three encysted abscesses in the liver.
- 301. A large abscess of right lobe and desminated abscess.
- 308-310. Perihepatic abscess—circumscribed and superficial.
- 311. Perforation of diaphragm and pericardium by perihepatic abscess.

In the Indian Journal of Medical and Physical Science, Vol I, p. 498, Webb in his list of pathological preparations in the Museum of the Calcutta Medical College mentions :—

Nos.

- 650. A section of inflamed liver from the vicinity of an abscess.
- 337. An irregular superficial abscess of the liver.
- 333. A cyst in a liver.
- 336. " " "
- 648. This preparation illustrates exceedingly well congestion of the liver followed by softening and suppuration.
- 779. A magnificent preparation of diffused abscess.
- 753. A fine specimen of encysted central abscess.
- 157. A magnificent specimen of ensysted abscess of the right lobe of the liver, forming a huge cavity which would contain a man's head.
- 555. Abscess burst into the lungs.
- 809. Three encysted abscesses.
- 885. In this remarkable preparation we observe first an old abscess which has strong adhesions to the false ribs on the right side. These adhesions give it the appearance of an aneurismal cyst projecting on the right of the gall-bladder. This old abscess was empty containing only a little yellow fluid, mixed with flocculence. It is lined by a sort of mucous membrane. This abscess had been evacuated by puncture, as marked by the glass-rod. Another of more recent date, and not

larger than an orange; is seen to have opened into the duodenum, just below the pylorus, immediately to the left of the gall bladder; to which the duodenum is seen to be intimately adherent. Another abscess formed in the centre of the left lobe, and then extended upwards, causing absorption of all the structures till it reached the diaphragm. Here it adhered strongly, and seems to have directed its course as if to open at one point into the pericardium, for it has perforated the diaphragm. The pericardium, however, and lungs, though both strongly matted to the diaphragm escaped. The abscess directed its course, downwards, opening into the stomach, about three inches below its cardiac orifice. The ulceration has proceeded through the mucous coat but the outer cellular coat, of the stomach, reduced to a sloughy state, hangs about the opening like a valve.

Thus it would be seen that the chief complications of liver abscess are peritonitis, pleurisy, pericarditis, pneumonia and abscess of the lung. The cerebral abscess is occasionally met with as a complication of liver abscess, but amæbæ are not found in the pus of these abscesses. Kartulis has reported brain abscesses in 3 per cent. of his liver abscess cases. Jürgens had two cases of thrombosis of the femoral vein, in one of which amputation of the leg was necessary. Abscess of the spleen may sometimes occur. Hepatopulmonary abscess is a somewhat rare complication:—

Observers.	Number of cases.	Abscess of lung.
Harris	95	3
Fletcher	119	9-three of these ruptured into pleural cavity.
Strong	100	1; and 2 had empyæma.

Bradshaw¹ relates a case complicated with ascites, a rare condition which may confuse the diagnosis. "Suppurative hepatitis is likewise occasionally found complicated with induration and cirrhosis of the liver (Morehead), tubercle of the lungs, chronic ulcer of the stomach (Rouis), chronic nephritis (Cambay), &c."²

The chief complications are :—

1. *Cerebral abscess*: Godlee³ reports a case. Amœba may or may not be present.

2. *Strangulation of the small intestine*:—Rogers⁴ reports a case in which as a result of firm adhesions, fatal strangulation of the small intestine occurred.

3. *Compression of bile-ducts and traction on pylorus*:—Godlee⁵ is of opinion that from contraction, after successful operation, of the adhesions on the under-surface of the liver round the portal fissure, bile-ducts may be compressed producing jaundice, or colon or pylorus pulled, producing dilatation of the stomach.

4. *Thrombosis of the inferior vena cava*:—It is a rare

¹ Bradshaw. T. R. (Jan. 1908), "A clinical lecture on Tropical Abscess of the Liver" *Lancet*. vol. I. P. 146.

² Frerichs. *Diseases of the liver*, Vol. II, P. 140 *Syd. Soc. Trans.*

³ *Med. Chirrug. Trans.* vol. LXXXV, P. 119, 1902.

⁴ B. M. J. 1903, vol. I, P. 1316.

⁵ Rolleston's *Diseases of Liver*, P. 147.

complication. Rolleston¹ mentions a case under treatment of Dr. Ewart.

5. *Escape of bile from fistula of the operation wound*:—It is not uncommon. But cases like those reported by Godlee² where all the bile passed out in this way are surely rare.

6. *Lardaceous disease*—may occur as the result of continued discharge of pus.

7. *Secondary inflammation and suppuration*:—Apart from rupture, it may give rise to secondary inflammation and suppuration, especially in the thorax.

(1) Simple serous pleurisy (right).—Through diaphragm.

(2) Empyæma of right side through diaphragm; may be encysted between the lobes of lung as in Duplant's case³.

(3) Pleurisy and adhesions between the base of the lung and diaphragm.

(4) Pneumonic consolidation at base, containing an abscess.

(5) Rupture into lung and pleura and the formation of a broncho-biliary fistula.

¹ Med. Chirrug. Trans. vol. LXXXV, P. 121.

² Med. Chirrug. Trans. LXXXV, P. 123.

³ Duplant: Lyon Medical. Jan. 26, 1902.

The following table will show the percentage of Mortality. mortality of liver abscess as noted by the various authorities.

Authority.	Country.	No. of Cases.	Recovery.	Died.	Mortality. Per cent.
Ramirez ¹		11	1	10	90
Rouis	Algeria	203	{ 39 2 improved }	162	80
Castro	Egypt	125			72.5 to 76
		208	115	93	57.7
Waring	India	81	15	66	81.481
		(operated)-			
Osler	United States	50
Indian army	India.				
(1891-94 & 1901-03)	522	236	286	54.7
Med. Chir. Soc.	Alexandria	72		58	80.5
Dujardin-Beaumetz		has fallen from 80 to 32 per cent

In connection with the very small proportion of recovery Waring remarks that even "this proportion small as it is, I fear is overrated, as it is a practice with some medical men, to give prominence to successful cases, and to bury in oblivion those which prove unsuccessful. It is a practice alike unphilosophical and culpable but easily comprehensible, no one likes to be the herald of his own failures". It is worthy of note that in none of these 15 cases of recovery recorded by Waring does the hepatic affection appear to have been combined with dysentery. In spite of the above remark, there is no doubt that the death rate has been

¹ Du Traitment des Abces du foie, &c. 1867.

lowered of late years, owing to greater fearlessness with which surgeons now treat the disease. This will be apparent from the statistics of operations, to be dealt with afterwards.

The cause of death differs in different circumstances.

This would be evident from the following
Cause of death. tables :—

Rouis—162 fatal cases—

Severity of the local disease or dysentery	125
Bursting of the abscess into peritoneal cavity	12
Bursting of the abscess into pleura	11
Gangrene of abscess wall	3
Peritonitis	3
Pneumonia from effusion of liver pus into lung	3
Rupture of adhesions	2
Pneumonia	2
Rupture into pericardium	1

162

Waring—51 cases—

The presence of other abscesses in liver besides those opened	...	19
A combination with dysentery	...	17
Gangrene or sloughing condition of the abscess wall	...	4
Abscesses communicating with the lung	...	2
„ „ „ colon	...	1
„ „ „ pericardium	...	1
Two other abscesses opening spontaneously	...	1
Another abscess bursting into abdominal cavity after operation	...	1
Impervious state of the hepatic duct, delirium	...	1
Escape of matter through the puncture into the abdominal cavity causing excessive inflammation	...	3
Abscess making its way by ulcerative absorption through the coats of the stomach	...	1
Hæmorrhage into the sac of the abscess	...	1

But we see that the majority of patients with hepatic abscess die of exhaustion from hectic rise of temperature or from diarrhoea or dysentery either during the course of the disease, or after it has burst naturally or opened by the surgeon. We have noted once before, that the supervention of diarrhoea or dysentery was often the fatal symptom, after operations of liver abscess performed by us.

The symptoms of liver abscess are generally described in works of medicine as being much more uniform than they really are. It would seem from the picturesque descriptions given there, that nothing could be easier than the diagnosis of liver abscess. On the contrary, it is far otherwise in actual practice; none of the various symptoms can be identified as pathognomonic of the disease. Even the experienced surgeons often confess their inability in many cases. To diagnose it properly, we should consider the circumstances under which the disease arises; and no hard and fast rule can be laid down to guide the inexperienced. It should always be borne in mind, that such a grave disease may for a long time, be unattended with any of the urgent symptoms noted before. Rouis found 13 per cent. of his cases to be latent. The symptomatology of his cases has been classified as follows :—

		Number in 143 cases.	Number per cent.	
Hepatitis Acuta.	{ Accompanied by all the peculiar symptoms.	11	8	Perfect symptoms.
	{ By only a portion of these symptoms.	15	10	79 per cent. Imperfect symptoms.
Hepatitis Subacuta	{ A well defined commencement; obscure progress during the middle period; well marked symptoms at the commencement of suppuration.	6	4	
Hepatitis Chronica.	{ Undefined symptoms or none at all.	62	44	
	{ Undefined symptoms during the entire progress of the disease.	30	21	13 per cent. symptoms latent.
	{ Progress marked, or latent ...	19	13	

Commonly the following diseases may be mistaken in diagnosis:—Hepatitis, softening syphilitic gummata, pylephlebitis, suppurating hydatids, gall-stones and inflammation of the gall-bladder,¹ subphrenic abscess, parietal abscess, pleurisy, encysted empyæma, pyelitis of the right kidney, pernicious anæmia, leucocythæmia, basal pneumonia, malarious fever, abscess of left lung, splenic tumour, &c. Again marked pulsation may be observed in connection with an hepatic abscess presenting in the

¹ Two cases where through mistake in diagnosis, the gall bladder was punctured and the patient died, are recorded by Dr. Stokes. Dublin Hospital Reports, No. V.

epigastrium, conducted from the aorta, and simulating an aneurism.

Bennet ¹ has described a case, who had dysentery before and which proved fatal when a phantom tumour was operated on.

It has been mistaken for acute hepatitis occurring in infectious liver ² and in obscure forms of septicæmic cases and in typhoid fever ³.

Hepatic gumma have been operated upon under the idea of an hepatic abscess.

Hatch ⁴ and Powell ⁵ relate cases where abdomen was tapped, mistaking liver abscess for ascites. Josserand ⁶ mentions that a case suffering from pulmonary tuberculosis, hectic rise of temperature and fatty liver was mistaken for liver abscess. Rutherford ⁷ mentions a case in which an extensive abscess of the right kidney simulated abscess of the liver.

¹ Lancet 1902. Vol. I p. 3.

² Remlinger : La Presse. Médicale 1903 p. 86.

³ Bozzolo : Rivista Critica de Clinica Medica. March 1902.
H. Jones B. M. J. 1897. Vol. II p. 1581.

⁴ Hatch : I. M. G. August 1898.

⁵ Powell I. M. G. February 1898.

⁶ Josserand, Jauru de Med. July 25, p. 98.

⁷ Annals of Naval and Military Surgery, from Army Medical Report. 1861.

Thus we see any pathological condition around and in the hepatic area may simulate abscess of the liver. We would rest contented by tabulating the differential diagnosis in a few cases.

LIVER ABSCESS.

MALARIOUS FEVER.

- | | |
|--|-----------------------------------|
| 1. History of alcoholism or dysentery. | No such history. |
| 2. Polymorphonuclear leucocytosis characteristic (may be present in other diseases). | Absent. |
| 3. Amæba in pus. | Malarial parasites in blood. |
| 4. Irregular intermittent fever—Evening rise. | Quartan or tertian periodicity. |
| Quinine useless. | Fever earlier in the day. |
| 5. Enlargement of spleen rare. | Quinine specific. |
| Enlargement of liver localised or tumour like. | It is the rule. |
| | General enlargement of the liver. |

LIVER ABSCESS.

HYDATID CYSTS.

- | | |
|---|---|
| 1. History of hepatitis, dysentery or alcohol. | No such history. |
| 2. Growth rapid and painful. | Slow and painless. |
| 3. Constitutional symptoms often marked. | Not so: but the pressure symptoms are marked. |
| 4. Amæba in pus. | Hooklets in pus. |
| 5. Changes in size occur corresponding to different stages. | No such change of size of the tumour. |
| 6. Septicæmic fever. Increase of temperature. | No fever, no rise of temperature. |
| 7. Hard tumour with soft summit. | Purring tumour. |
| 8. Common in tropics. | Common in Australia and Iceland. |
| 9. Nutrition impaired. | Nil. |

LIVER ABSCESS.

INFLAMED AND DISTENDED GALL-BLADDER.

- | | |
|---|--|
| 1. History of dysentery or alcohol often preceded by induration. | History of colic. No induration before. |
| 2. Tumour often diffused. | Pear shaped or globular. |
| 3. Fixed. | Mobile. |
| 4. Adhesions the rule. | Scarce. |
| 5. Edema present. | Nil. |
| 6. The diffused tumour is soft and fluctuating at the summit, while the base is hard and resisting. | Constantly soft and fluctuating. The tumour is circumscribed and hard and equally resisting in every part. |
| 7. Fever present. | No fever. |
| 8. Pain diffused. | Pain limited to pyriform body. |
| 9. Liver enlarged. | Liver normal. |

LIVER ABSCESS.

PYLEPHLEBITIC ABSCESS.

- | | |
|----------------------------------|--|
| 1. Traceable to dysentery. | Traceable to suppurative focus in the portal area. |
| 3. Non-uniform enlargement. | Uniform and tender enlargement. |
| 3. Course often chronic. | Acute course. |
| 4. Constitutional symptoms slow. | Rapid. |
| 5. Splenic enlargement rare. | Common. |
| 6. Jaundice absent. | Jaundice marked. |

LIVER ABSCESS.

CANCER.

- | | |
|---|---|
| 1. Liver may or may not be enlarged. | Slow enlargement with pain. |
| 2. Smooth and elastic. Fluctuation sometimes. | Nodular state of liver. No fluctuation. |
| 3. Ascites rare. | Common. |
| 4. Rigor, fever and sweats diagnostic. | Absent. |
| 5. Occurs at any period of life. | After middle age. |
| 6. History of dysentery or alcohol. | No such history. |
| 7. Duration short. | Duration long. |

LIVER ABSCESS BURSTING INTO LUNG.

EMPHYEMA.

- | | |
|--------------------------------------|--------------------------------------|
| 1. Anchovy-sauce like colour of pus. | Ordinary pus. |
| Amæba present. | No amæba. |
| Presence of hepatic tissue or bile. | Nil. |
| 2. Disturbance in hepatic functions. | The effusion is preceded by pain and |
| Fluctuation over the liver. | accompanied by dyspnoea. |
| 3. History of dysentery or alcohol. | No such history. |

LIVER ABSCESS.

DEEP PARIETAL ABSCESS.

- | | |
|---|---|
| 1. Marked constitutional symptoms. | Slight. |
| 2. History of dysentery or alcohol. | Nil. |
| 3. The tumour corresponds to the hepatic area. | The tumour does not correspond to the area of enlarged liver, |
| 4. Integument not very hard or tight. | There is greatest amount of hardness and tightness of the skin. |
| 5. The tumour follows the respiratory movements unless there be firm adhesions. | The up and down movement is absent. |

LIVER ABSCESS.

PYÆMIC ABSCESS OF THE LIVER.

- | | |
|--|---|
| 1. Smooth and elastic enlargement. | Moderate uniform enlargement. |
| Abscess single, fluctuates if superficial. | Abscess multiple. No fluctuation. |
| 2. History of dysentery or alcohol. | History of injury and suppurative processes in other sites. |
| Amæba in pus. | Nil. |
| 3. Jaundice absent. | Present in $\frac{4}{5}$ th of cases. |
| 4. Course slow—3 weeks to as many years. | Rapid—3 weeks to 3 months. Symptoms of blood-poisoning. |
| 5. Tropical. | Occurs everywhere |
| 6. Serious but not always fatal. | Much the more serious and fatal. |
| Recovery not uncommon. | Recovery rare. |
| 7. Various terminate | Terminations of pyæmia. |
| 8. Amenable to surgical operation. | Operation worse than useless. |

FEVER OF LIVER ABSCESS.

1. Rigor not constant, often fever comes with chill. Sweating marked during sleep.
2. Jaundice absent.
3. Fever remittent at first, later, intermittent.
4. Wasting marked.

INTERMITTENT HEPATIC FEVER WITH GALL-STONES.

- Fever with rigors and sweats occurs regularly but often after long intervals.
- Jaundice deepens after the fever.
- Entire apyrexia in the intervals.
- Not marked.

LIVER ABSCESS.

1. The site, size and form of the tumour correspond to the hepatic area.
2. On percussion—dull area of a solid tumour.
3. Swelling is unchanged by change of posture.
4. Under chloroform the swelling appears more clearly.

MUSCULAR CONTRACTION ESP. OF UPPER SEGMENT OF RIGHT RECTUS.

- The site size and form correspond to the division of the rectus muscle.
- The sound is more clear and tympanic.
- On sitting the swelling contracts and becomes thicker.
- Then the tumour disappears.

LIVER ABSCESS.

1. Liver enlarged.
2. Circumscribed hardness.
3. General derangements marked.
4. Progress rapid.

PERI-HEPATITIS.

- Nil.
- Absent.
- Insignificant.
- Much more rapid.

LIVER ABSCESS.

1. Cause:—Dysentery or alcohol.

SUBPHRENIC ABSCESS.

- Causes: may follow hepatic abscess, suppurating hydatid cyst, suppurative cholecystitis (1), perforation of gastric or duodenal ulcer (2), appendicitis (3), calculous and tuberculous diseases of kidney, suppuration in spleen and malignant diseases of large intestine.

Symptoms of pyopneumothorax absent. If (1): We get symptoms of intrahepatic abscess.

If (2): Subphrenic Pyopneumothorax: Resonance on percussion; a bell note; succussion.

If (3): History of recent appendicitis; signs of appendicular mischief in the right iliac fossa;

Tendency to bulge into right loin.¹

LIVER ABSCESS.

1. Fever the rule.
2. Traumatic rarely; history of dysentery or alcohol.
3. Tumour often ill-defined.

PANCREATIC CYST.

No Fever.

History of blow.

Definite tumour of large size in the left hypochondrium.

LIVER ABSCESS.

1. Suppuration above umbilicus.
2. Dulness marked.
Liver enlarged.
3. The needle moves with respiration.

SUPPURATION IN THE RECTUS.

Suppuration in the sheath—rare—after typhoid and occurs below the umbilicus.

If above umbilicus: no complete dulness over the swelling; no enlargement of liver.

Miodeldorff's method—a needle in an abscess of abdominal wall is stationary.

LIVER ABSCESS.

1. Generally liver is enlarged.
2. Polymorphonuclear leucocytosis.
3. Glands not affected.

LYPHADENOMA.²

Liver not much enlarged.
No leucocytosis.
Superficial gland affected.

Roughly speaking, we may be sure of tropical abscess of the liver if there be history of malaria, dysentery and

¹ See Elsberg: Subphrenic Abscess after Appendicitis. *Annals of Surgery* Vol. XXXIV. P. 720, 1901.

² See Rolleston's case in *Diseases of Liver* p. 152.

alcoholism, if there be presence of swelling, of pain and tenderness in the hepatic region, with or without fluctuation; of muddy complexion, the anxious expression, and of the fever preceded by rigor; and lastly if, by aspiration pus be discovered and amæba be found in it under the microscope.

Skiagraphy: "In health the shadow cast on the screen, shows that the right leaflet of the diaphragm is $1\frac{1}{2}$ inches above the level of the left leaflet and is constantly moving with respiration. In cases of abscess, it is displaced upwards and does not move" (Rolleston).

The prognosis of liver abscess is generally very unfavourable. The percentage of mortality varies. If every cases of hepatitis is included, then the mortality is very small. The statistics show a mortality of 30 per cent. If we take only cases of evident pus formation, the mortality is very high indeed.—70 to 80 per cent. In operated cases the mortality is generally 40 to 50 per cent., but it has been much reduced in recent times. In Solonoff's collection of 1094 cases, the mortality is 30 per cent.

The prognosis depends upon the following circumstances :—

- (1) The coexistence of active dysentery, a history of

¹ Solonoff. B. M. J. 1903, Vol. I, P. 262.

alcoholism, bad health, continued discharge from abscess leading to lardaceous disease, albuminuria, œdema of feet and diarrhoea are grave complications. The influence of dysentery upon the progress and the mode of termination of liver abscess can be shown as follows¹ :—

Cases.	Dysentery.	Opened externally.	Recovery.
24	Absent	19 or 80 per cent.	14 or 60 per cent
		5...abdominal wall	... 4
		8...Bronchi	... 6
		6...Intestines	... 4
118	Present	59 or 50 per cent.	25 or 29 per cent.
		29...Abdominal wall	... 13
		22...Bronchi	... 9
		8...Intestines	... 3

Prognosis varies in the different modes of spontaneous termination :—

Anterior wall of the abdomen	...	favourable.
Bowels
Bronchus „ but course tedious.
Pleura fatal.
Stomach fatal.
Pericardium „
Peritoneum „
Inf. vena cava „
Ureter „
Loin Unfavourable.

(2) The number of abscesses also determines the prognosis. Multiple abscesses are often fatal. This is true, however, as a general statement of facts, but exceptions

¹ Frerich's Disease of the Liver, Vol. II, P. 136-37 Syd. Soc. 25.

undoubtedly occur. Budd mentions that Lawson practised as a surgeon for 10 years after an attack of hepatitis, which left behind several small abscesses. Cassimir Bronssais states that in the autopsy of a man, who had survived an attack of dysentery and hepatitis previously, four cicatrices were found in the liver. In Madras General Hospital 1893-1902, of the 154 cases, 21 were multiple and they all died. Of the remaining 133 cases, 53 died but we are not sure that they were all single, as we have not definite remarks of the surgeons. In Niblock's 29 cases, 5 were multiple and all died; and amongst the remaining 24 there were 6 deaths or 25 per cent.

Prognosis also depends upon the nature of the abscess. Lafleur¹ thinks the amœbic abscesses to be more fatal, but Manson² denies it.

(3) Rupture into pericardium, pleura and peritoneal cavity is always fatal. There is always a chance of recovery if the pus finds for itself an exit, as through bowels or lungs.

Observer.	Cases in which abscess discharged through the right lung.	Recovery.
Bartholow		$\frac{1}{2}$ of his cases.
Roux.	30	15
De Castro ³	25	19 or 76 per cent.

¹ Lafleur. Albutt's System of Medicine Vol. IV, 1st Ed., P. 168.

² Manson. Tropical Diseases, 1903, P. 457.

³ De Castro. Des Abscess du foie des pays chauds, Paris 1870.

Observer.	Cases in which abscess discharged through the right lung.	Recovery.
Morehead	5	3
Stovell	16	9
Laffeur } Godlee }	...	Unfavourable.

The most favourable mode of exit is through the lungs. The discharge of pus may continue for months and yet recovery may take place. Discharge of pus externally is said to be another favourable termination but it is rare. Maclean confirms Morehead in the fact that the abscesses which open at the ensiform cartilage have better chances of cure than those pointing in the right hypochondrium or in an intercostal space. This is due to the collection of pus in the former case being seated in the left lobe of the liver, and therefore smaller. Rupture into colon is usually followed by recovery.

(4) Prognosis depends upon the early or late surgical operation. Since hepatic abscesses have been treated by free incision and drainage, the mortality has fallen from 80 to 32 per cent (Dujardin-Beaumetz). By operation, the best results are obtained in cases of single abscess situated near the surface and not complicated with dysentery. In Niblock's cases, "all abscesses containing less than 20 ounces of pus at the time of operation recovered". But the statistics of the results of making artificial opening are by no means uniform and encouraging. There

can be no doubt that the operation often prolongs life. Once the presence of pus is diagnosed, delay in surgical operation cannot be justified, and the postponement of the operation for even a single day may lead to a perforation of some neighbouring organs, and the results may be disastrous.

(5) In private cases, the prognosis is better than what happens in hospital. Morehead¹ thinks that about 70 per cent. of the former and 50 per cent. of the latter recover.

Hatch² found that in some years, the mortality in hospital was 90 per cent., while in private practice it was about 20 per cent. only.

As regards the health of a patient who has recovered from an attack of liver abscess there is reason to believe that he will regain his usual standard of health. Cases are not uncommon where individuals have lived 30 or 40 years after the occurrence of abscess, and Macpherson³ knew a man of 75, who had had abscess 45 years before.

Thus the question suggests itself for solution. "Can a liver, the large part of which is rendered functionless by disease, perform its physiological functions as usual"?

To this question it is difficult to give an answer offhand, unless it be from the recorded evidence of observers

¹ Morehead. B. M. J. 1899. I. P. 1032.

² Lancet 1902 Vol. II. P. 1543.

³ Quain's Dictionary of medicine, Part I. P. 831.

who knew patients in good health, many years after the disease. Cantlie remarks¹: "When one part of the liver is rendered functionless or destroyed, the remaining portion becomes hypertrophied, and takes up the work of the diseased portion". By observations and experiments he claims to have proved "that there are two *sides* to the liver, each equal in bulk, and each half presided over by an equal-sized branch of the portal vein, hepatic artery, and hepatic duct. In fact there is a right and left liver in juxtaposition, equal in weight, but having separate vascular and biliary circulation. Disease of one side of the liver need not, and very seldom does, involve the other; moreover when one side is destroyed the other may take up its functions. There is nothing unique in this, for we have the example of one kidney executing the work of both when one is removed. A liver, therefore, in which disease is confined to one side may come in time to fulfil the function of the whole; and given time to develop, the compensatory power is so marked that a person may recover completely with only half a liver left."

Recurrence:—After apparent recovery, there is danger of recurrence. So Marshall² advises that no patient should return to tropics, until after 2 years and better, if not at all.

¹ Encyclopædia Medica, vol. 7. P. 28.

² Marshall, B. M. J. 1899, vol. I, P. 1387.

CHAPTER X.

Prophylaxis: improved sanitation; The presuppurative stage of liver abscess; its diagnosis by leucocytosis; the value of leucocytosis; Ipecacuanha treatment; In what stage is it useful? Medical treatment of liver abscess. History of Ipecac treatment, its disadvantages; its mode of administration.

There can be no doubt that the tropical abscess of the liver is a preventible disease. No matter on what does it depend, the faulty condition can always be rectified. This can be inferred from the fall in mortality amongst the English army in India.¹

1870-79 : mortality 2·19 per thousand.

1891-1900: „ 1·24 „ „

Locality.	Year.	Army.	Strength.	Admission.	Rate.	Death.	Rate. per cent.
India	{ 1870-79	European	50	...	2·19
	{ 1880-89	...	59,796	15,825	26·8	803	1·34
Bengal	{ 1870-79	47	...	2·04
	{ 1880-89	25·8	...	1·25
Madras	{ 1870-79	67	...	3·16
	{ 1880-89	36·9	...	1·93
Bombay	{ 1870-79	41	...	1·71
	{ 1880-89	21·4	...	1·01

¹ Davidson's Diseases of Warm Climates p. 648.

HEPATITIS.

Country.	Year.	Admission rate.		Death rate.	
		L.A. without dysentery.	L.A. with dysentery.	L.A. without dysentery.	L.A. with dysentery.
India	1889	...	1·81	·20	1·05
Bengal	1·65	·35	·94
Madras	1·84	·07	1·39
Bombay	2·17		1·08

Army.	Country.	Strength.	Year.	Admission.	Per cent.	Death.	Per cent.	
European	India	{	69017	1889	{ 125 -	1·81	73	1·05
			67823	1890	{ 14 +	·20	13	·18
	Bengal	{	1889	{	97 -	1·42	43	·83
					24 +	·35	28	·41
			1890	{	71 -	1·65	45	·94
					13 +	·35	12	·27
	Madras	{	1889	{	50 -	1·29	23	·55
					24 +	·58	26	·62 ¹
			1890	{	26 -	1·84	19	1·39
					1 +	·07	1	·07
Bombay	{	1889	{	22 -	1·60	11	·80	
				+			·14	
		1890	{	28 -	2·17	14	1·08	
				+	0		0	
Native	{	128642	1889	{	25 -	1·95	9	·71
					0			0
	{	127746	1890	{				
	{	128642	1889	{			6	·05
							4	·03
	{	127746	1890	{				0·2

In the above table, the sign + represents cases of liver abscess with dysentery, and the sign - stands for cases without it. The death ratio of the European army from hepatic abscess was 35 times higher in 1890 and 25 times higher in 1888 and 1889 than that of the native army.

This improvement is due to scientific methods of sanitation, ventilated barracks and more temperate habits of the soldiers. The idiopathic abscess is less frequent; and

¹ The figures include mortality out of hospital.

the amæbic infection requires prevetion. To attain this end, supply of pure water is the most important factor.

The amæbic dysentery should be prevented by:

- (1) Disinfection and safe disposal of stools.
- (2) Prevention of pollution of soil and water—the substitution of water-carriage system for privies and cesspools.
- (3) Supply of pure water and its safe storage.
- (4) Thorough draining of the soil.
- (5) The boiling of water before use.
- (6) The cleanly cultivation and scrupulous washing of raw vegetables.
- (7) And easily digested diet and avoidance of fatigue, exposure and excesses.

If a man becomes dysenteric, he should be cured by ipecacuanha and other medicines; if he be suffering from malaria, re-infection may be prevented by the judicious administration of quinine. Exposure to cold should be avoided in tropical climate. And last of all, though not the least in importance, ardent spirits should be shunned by all as poison in the tropics. Excesses at the table, much animal food, rich sauces, hot curries, and pastry—can not be long continued and should be avoided by the European. He who is moderate in all things is the safest. In Waring's statistics¹, liver-abscess is rare amongst teetotallers.² The officers, long subject to hepatic disorders,

¹ I. A. No. vi. p. 484.

² Influence of Habits of Life in the Causation of Liver Abscess. p. 617.

when imprisoned by Hyder Ali, lived on a scanty diet and returned in good health. Murray's case¹, an officer, who being ship-wrecked on a desert island lived exclusively on eggs, was completely cured of his hepatitis. But the animal diet need not be too much curtailed as some cannot digest vegetable food. Lighter wines in moderation may be allowed. Acute hepatic complaints are unknown amongst the French inhabitants of Pondicherry², as they live principally on vegetable food and curries, and drink the light wines of France in moderation.

The presuppurative stage of amœbic hepatitis:—
Major Rogers of Calcutta in 1905 discussed the value of leucocytosis in acute hepatitis, and concluded that "its presence in a marked degree was generally an indication that suppuration had already taken place; but in the slighter degrees, it may be present, in acute hepatitis without actual suppuration or that this early stage of amœbic hepatitis may sometimes be cured, and suppuration prevented, by the administration of large doses of ipecacuanha." He has shown in his "Fevers in the Tropics" by careful research that the disease may frequently be recognized by the blood changes when

¹ Bombay Medical and Physical Transactions, Vol. i.

² Madras Medical Topography, Centre Division, p. 66;

in a stage which admits of cure. "Thus the patient is prevented from drifting on into the much more serious suppurative stage, now so commonly their fate. Further these (his cases) constitute a distinct class of fever, usually of a chronic intermittent type, sometimes with no very definite symptom of hepatitis, rarely with any dysentery. They may be recognized or at least strongly suspected by the presence of a moderate degree of leucocytosis, generally of the type which I have described as common in amœbic abscess of the liver, namely one in which the proportion of polynuclears is either normal or only slightly in excess. Further—and this is the most important practical point—this kind of fever yields rapidly to large doses of ipecacuanha in the absence of symptoms of dysentery, or even of hepatitis, and the formation of tropical abscess of the liver is thus prevented." If this fascinating description be found out to be true by other competent observers, no doubt it would be a great achievement in medical science.

The existence of marked leucocytosis—from 3000 to 5000—has led to the detection of hepatic abscess in patients suffering from dysentery (Boinet¹) or malaria (Rogers²).

¹ Soc. de Biolog. Path. Dic. 29. 1900.

² B. M. J. 1902, I. 831 and II. 844.

Before accepting the observations of Rogers as proved, we have to solve the following questions:—

1. Is the leucocytosis, a special feature of amœbic hepatitis only, or is it found in other diseases?

Now let us study the leucocytosis of Rogers' cases. They are as follows:—

No.	R. B. C.	W. B. C.	Ratio.	Polynuclear.	Lymphocytes.	Large Mononuclear.	Eosinophile.
1	3600000	15250	1-236	83	13	4	0
2	Leucocytosis		...	78	16	2	4
3	"		...	79	12	6	3
4	3090000	24000	1-129	78	15	7	0
5	3610000	285000	1-126	76	21	3	0
6	3960000	9500	1-416	78	8	3	2
7	4820000	21000	1-230	78	15	5	2
8	4600000	17750	1-233	87	7	5	1
9	Leucocytosis		...	80	15	4	1
10	5380000	17000	1-316	76	16	6	2
11	4610000	9500	1-517	79	16	4	1
12	4290000	15250	1-272	81	11	7	1
13	Leucocytosis	
14	4660000	20750	1-225
15	3340000	17250	1-193	74	22	3	1

To verify these results we examined the blood of patients in whom liver abscess was suspected but not definitely diagnosed, and who afterwards developed abscess of the liver. So this stage can safely be taken as the presuppura-

tive stage of liver abscess. The patients were all natives, and we suspected abscess of the liver because of their intemperate habits. The type of leucocytosis present in them does by no means correspond to the type of Rogers' cases.

No.	R. B. C.	W B. C.	Ratio.	Polynuclear.	Lymphocytes.	Large Mononu clear.	Eosinophile.
1	4530000	23000	1-197	90	7	2	1
2	4840000	16200	1-293	70	13	5	2
3	3900000	12800	1-305	96	2	1	1
4	5240000	18000	1-403	68	22	7	3
5	4430000	17000	1-249	74	16	6	4

The characteristic leucocytosis in which the proportion of polynuclears is normal or only slightly in excess is absent in the above cases. The percentage of polynuclears varied from 68 to 96. Again Strong says that "there is usually a leucocytosis from 15000 to 40000 in which polymorphonuclears are increased. However the leucocyte count may be normal¹." Moreover if we compare the leucocytosis in epidemic dropsy we shall find that the figures nearly approach those of Rogers' cases. The

¹ Strong in Osler's System of Medicine. Vol. I., P. 516.

following are the figures of leucocytosis in epidemic dropsy counted by Rogers himself¹ :—

No.	R. B. C.	W. B. C.	Ratio.	Polynuclear.	Lymphocytes.	Large Mononuclear.	Eosinophile.
1	3490000	10375	1.329	71.6	20.0	7.2	1.2
2	2490000	7250	1.340	71.2	22.0	6.8	0.0

Again Rolleston says that leucocytosis, though highly suggestive of hepatic abscess, is by no means found in all cases. When septic absorption is prevented by a thick capsule and in amœbic cases, there may be no leucocytosis.²

Thus we see that the leucocytosis is not a special feature of abscess of the liver. The same type of leucocytosis in which the proportion of polynuclear is either normal or slightly in excess is found in other forms of suppurative processes, and sometimes in inflammatory processes which do not end in suppuration. Leucocytosis is also seen in the later stages of diseases involving destruction of liver tissue (*e. g.* cirrhosis).³

¹ Discussion on Epidemic Dropsy in Asiatic Society Bengal. I. M. G. vol. xliii, No. 405, 1908.

² Rolleston. Diseases of Liver, p. 139.

³ Richard C. Cabot in Osler and Macrae's System of Medicine Vol. iv, p. 608. 1908.

Boinet¹ thinks that leucocytosis when present, is constant and does not appear and disappear as it does in intermittent hepatic fever; but other observers affirm that leucocytosis is by no means a constant feature in liver abscess; "when septic absorption is prevented by a thick capsule, and in amœbic cases there may be no leucocytosis" (Rolleston). It was absent in 3 out of 5 cases (Osler)². It was absent in Cabot's case³ and Rispal⁴ found slight leucocytosis (15,000) in 3 cases.

A very interesting case, illustrating the fallacy of accepting leucocytosis as the sure sign of liver abscess, was operated the other day in a hospital, at Calcutta. The patient's blood was examined by the bacteriologist of the hospital and it was pronounced to be a case of tropical abscess in the presuppurative stage. But on operation, it was found out to be a case of cirrhosis of the liver.

So we think that the leucocyte count does not help us much in arriving at a sure diagnosis of liver abscess. But it is of undoubted value in doubtful cases.

(2) Do the cases of hepatitis in which such leucocytosis is present, always end in suppuration if left to themselves?

¹ Boinet: Soc. de Biolog. Paris, Dic. 29, 1900.

² Osler: Med. News (N. Y) April 12, 1902, p. 673.

³ Cabot: Clinical Examination of the Blood p. 253.

⁴ Rispal: Compt. rend. Soc. de Biolog, 1991. p. 362.

We can not say that the cases mentioned by Rogers would have terminated in supuration if left to nature. There is no doubt that by the use of ipecacuanha, the fever accompanying the hepatitis has been checked in a remarkable way. But we can affirm so far and no further. It requires to be proved that in liver abscess only one peculiar type of leucocytosis occurs; and that the cases in which such a type is present, invariably end in supuration, if not treated by ipecacuanha.

(3) Are those cases of fever without dysentery or liver symptoms but having a marked leucocytosis, (group IV of Rogers' cases), really cases of amœbic hepatitis, because they were amenable to the ipecacuanha treatment?

We can not say with certainty that these cases would have terminated in abscess. There were no symptoms of liver disease or of dysentery. The only reason for calling them cases of liver abscess is the presence of the peculiar type of leucocytosis. Cases of liver abscess without any sign pointing to the liver, are possible; but generally we get some symptoms of the disease.

(4) Is it possible by medicinal means to check suppurative process already started in the liver substance?

Surely when supuration has once declared itself, we can not hope to check the process by any medicine. But

if it be possible to diagnose the presuppurative stage, then there is nothing inconceivable in the assumption that there may be medicaments which may check the process. And we may say with Burney Yeo¹ that "Assuming ipecacuanha to contain a principle which has the power of destroying the activity of the amœbæ coli, or their secretions, and supposing this principle to be soluble in the intestinal contents and to be carried to the liver through precisely the same channel as the toxic irritant, and supposing this irritant to be only of feeble virulence, there seems nothing irrational and impossible in the hypothesis that its infective potency may be stayed, and the pathological processes which might end in suppuration abated."

(5) What is the action of ipecacuanha on the liver?

Acting remotely on the liver, this drug is a direct cholagogue, increasing the secretion of bile. The action of ipecacuanha in large doses has been thus summarised by Ewart². "It produces all the benefits that have been ascribed to blood letting, without robbing the system of

¹ Yeo's Medical Treatment, Vol. II, P. 184.

² Ewart's Review of treatment of Tropical Diseases in Indian Annals of Medical Science, 1862-63, P. 403.

Sir J. Fayrer also prescribes ipecacuanha during the acute stage. [See Davidsons' Diseases of Warm Climate, p. 661].

one drop of blood; it produces all the advantages of mercurial and other purgatives, without their irritant action; it produces all the good results of antimonials and sudorifics, without any of their uncertainty; and it produces all the beneficial effects ascribed to opium, without masking, if not aggravating the disease, whilst the mischief is silently accumulating within. Thus, it may be said that we possess in ipecacuanha, a direct and safe antiphlogistic, a powerful sudorific, an unirritating purgative, a certain cholagogue, a pancreatic stimulant, and a harmless sedative to the heart and muscular fibres of large intestine." He too claims for the drug the merit of decreasing the frequency of liver abscess.

The supporters of the ipecacuanha treatment are not agreed as to the stage of the abscess in which it acts as a specific. Captain Murray¹ writes: "*It must be clearly understood that only cases in the presuppurative stage are referred to.*" I do not, for one moment, mean to imply that once an abscess has formed, ipecacuanha will be of any use whatever." Captain Greig² would extend its use even after evacuation of abscess: "Observations were made also, on a case, in which two amoebic abscesses in

In what stage
is Ipecac use-
ful?

¹ Murray. I. M. G. Vol. xliii. No. 4. 1908.

² Greig. Ibid. xlv. No. 1910.

the liver had been opened, and as the temperature still remained elevated, Pulv Ipecac in full doses was given, with the result that the temperature and leucocyte curves fell shortly afterwards to normal, and the patient recovered. In this case the drug appeared to have limited the amœbic infection and prevented a further extension in the liver." Lt.-Col. Nott¹ mentions a case in which ipecacuanha was not given before operation. "In this case some 17 or 18 ounces of pus were withdrawn by aspiration and quinine solution injected and Ipecacuanha which at first was badly taken, was given in large doses. Two further aspirations were made at intervals of 10 days, although the temperature was mostly normal, and further quantities of pus withdrawn. Eventually after taking Ipecacuanha for a long period recovery took place." Lt.-Col. Pilgrim² emphasised "the necessity of continuing the ipecac treatment for a considerable time after all the symptoms have disappeared." Thus we see that ipecac treatment is recommended in the various stages of the disease; and beneficial results were obtained in all cases. Cantlie³, on the other hand, questions the high hopes entertained by Indian physicians about the ipecac treatment

¹ Nott. Ibid.

² Pilgrim. Ibid.

³ Cantlie. Paper on the treatment of chronic Dysentery. Section of Tropical Medicine. September 1909.

and says : "The so-called presuppurative stage of hepatitis in the history of the formation of a liver abscess is a misnomer, and the exhibition of Ipecacuanha to control the hepatitis is not a rational explanation of the good claimed for Ipecacuanha in this respect. The prevention of the formation of liver abscess by the exhibition of Ipecacuanha, if it does thus serve, cannot be by controlling a general hepatitis, for it seldom exists as an antecedent to tropical abscess. The explanation, if such is feasible, may then be sought in the action of Ipecac. on the bowel contents, or upon the ulcerated surfaces; but this is a matter of mere speculation, for there is no surety on even credence that Ipecacuanha, more than castor oil, or Epsom salt, or milk, has any effect in preventing liver abscess." Sir Havlock Charles¹ remarks: "Sir P. Manson speaks highly about Ipecacuanha; it is a powerful drug, but on live amoebæ it is harmless; a theory is requisite to help its action. It undergoes change before absorption by the gut. Let it pass—will do as well as any other idea. I also have had recoveries, and many too, from threatened liver abscess, by the use of this drug." Pilgrim also admits of cases "in which cure was effected by rest in bed, suitable diet, the exhibition of salines, ammonium

¹ Charles. Discussion on his paper on Tropical Abscess of the Liver, July 1909, at the meeting of the British Medical Association.

chloride, quinine etc. and also local applications" but he observes that in many cases inspite of this treatment, the hepatitis has drifted on to abscess and claims for the ipecac. treatment that not a single case treated in this way drifted into liver abscess. Surely the testimony of so many observer in India can not easily be laid aside, as facts can not be ignored; but the trial has been given only for the last 3 years and it is premature to say that none of the cases developed liver abscess when treated by ipecac. in the early stage.

Thus we see that ipecacuanha would be of inestimable help in the treatment of liver abscess. But might not we suppose that ipecacuanha did good in Rogers' cases, because they were cases of hepatitis?

History of the ipecac. treatment. first employed as a remedy by Piso,¹ who brought it from the Brazils, and had given it in drachm doses, and in the form of infusion, but it was not until Helvetius, who had come from Holland to Paris, gave some of it, with a knowledge of its virtues, to the physician of Louis XIV., who employed it successfully in the case of the Dauphin, then dangerously attacked by dysentery, that it came

¹ De. Med. Brazil. Lib. ii. Amst. 1658.

generally into use. Marais,¹ and soon afterwards Sloane,² Heister, Vater, etc., further demonstrated its good effects. (Copland.³) Since that date, it has been employed by the highest authorities; and in modern times we find Annesley Twining, Ainslie, Gæddes, Mortimer, Ballingall, and Furguson, recording their testimony as to its value and efficacy in this disease" (Waring⁴). In 1783 Stephen Mathew recommended calomel and ipecac. pills in emetic doses in hepatitis. Next McLean of Netley urged its use as a preventive of tropical abscess of the liver. In 1885 Murchison confirmed McLean's views. Norman Chevers advocated large doses of ipecac. in hepatitis. Manson also speaks highly of it.

The chief disadvantage of the routine treatment by the drug in large doses is the unpleasant after-effects, the patients suffer from. It may be well to push the drug in the hospital patients but in private practice, such boldness will often be resented by the sick. The natives who suffer from liver abscess are generally of poor health and they can not as we have found it by experience, tolerate such big

Disadvantages
of ipecac-treat-
ment.

¹ Ergo. Dysent. Affect. Rad. Brasiliensis, Paris, 1690.

² Philosoph. Trans. No. cccxxviii.

³ Dict. Pract. Med. Vol. I.P. 731.

⁴ Waring, Practical Therapeutics, P. 298.

doses, even though the necessary precautions were taken by using keratinised or salol-coated capsules, or tannic acid, by the previous administration of opium or chloral, and by the hypodermic injection of emetine hydrochlor.

Again the question arises, as Lt.-Col. Harris puts it, "Whether Ipecac. should be given to produce vomiting as old writers advised or with precautions to prevent its occurrence." Dr. Arnold Caddy in conjunction with Dr. Kanthack had obtained good results with ipecac. sine emetine. Tull Walsh, on the other hand, had used emetine with mercury iodide which however forms an insoluble compound (Merek). Rogers thinks that the whole drug is more efficient than the sine emetina.

We may however conclude that the utility of the ipecacuanha treatment rests on the value of leucocytosis in liver abscess—a question which is far from being a settled point in pathology. But we think that there is no harm if it be laid down that "all cases of this disease should be treated with large and repeated doses of ipecacuanha, whenever there is any doubt remaining as to whether suppuration has already taken place or not, before any exploration of the liver is carried out, for this rule will certainly prevent some unnecessary surgical measures". It should, however, be clearly understood that no time should be lost, especially as Rogers has shown his cases

to be benefited by the treatment within 2 or 3 days, if there be any symptom of suppuration, in which case aspiration should at once be had recourse to. The abscess once diagnosed, no time should be lost upon any measure other than its opening and evacuation, even aspiration of pus will be useless. It is quite possible that the cases cited by Rogers were cases of desentery—latent or active—which kept the temperature of his patients high; and the fever subsided because the dysentery was cured by big doses of the drug.

Administration of Ipecacuanha :—Pilgrim describes in detail the mode of administration of the drug as follows :—

“ When not associated with loose stools and the bowels are on the contrary inclined to be costive, a mild mercurial purge is first given; otherwise the Ipecacuanha treatment is begun on the evening of admission or diagnosis of the disease. It is necessary that the patient should have nothing to eat or drink for at least 2 hours before and after the giving of Ipecacuanha. Twenty minutes before taking the Ipecacuanha I give 20 grs. of chloral, then in average cases I give 25 grs. of Ipecac; in severe cases in which the full influence of the drug is immediately required, I give 30 grs. for the first 3 or 4 nights, after that reducing it to 25 to 20 grs. gradually. I have occasionally given 40 grs., but I believe this is seldom

necessary, and the cases treated in this hospital have responded very favourably to 30 grs. doses and less. As a rule I find one dose daily suffices, but in severe cases I do not hesitate to give it night and morning, and also in cases not apparently severe but in which the leucocytosis does not rapidly reduce. The Ipecacuanha is given in keratine capsules 5 grs. in each, and I am quite sure that given in this way the Ipecacuanha treatment is robbed of half its horrors—given in capsules, both the nausea and vomiting are very greatly reduced—many patients do not vomit at all, but only suffer for a short time from nausea, while some few are absolutely free from any unpleasant or abnormal sensation; it is a question of whether the capsule breaks or comes undone before it has passed through the pylorus. * * * After swallowing the Ipecac. the patient is enjoined to lie absolutely still in bed when under the influence of chloral, he usually soon drops to sleep, and if he wakes up 2 or 3 hours later, feeling uncomfortable, the drug will at all events have largely exerted its influence”.

Lt.-Col. Calvert says: “More than 20 years ago I received the information, I believe, from the late Surgeon-General McLean—so the idea hails from Madras—that by combining tannic acid with Ipecacuanha, vomiting may be prevented; experience has shown the truth of this

statement. I mix 20 or 30 grs. of powdered Ipecacuanha radix with 10 grs. of tannic acid, flavoured with a few drops of oleum anisi and roll loosely into 5 grs. pills. Then the patient is directed to swallow quickly with as little water as is possible and then lie prone. There may be some nausea, but in nine cases out of ten, if properly carried out, there is no vomiting."

As regards the use of a mustard plaster, Pilgrim thinks "it a mistake to put on mustard plasters or other local applications to the stomach, which only attract attention to that organ, and interferes with the drowsy feeling which begins to steal over the patient ending soon in sleep".

CHAPTER XI.

Treatment : internal medicine, bleeding, leeches, ice. Obsolete operations—caustic, thermo-cautery, puncture. Expectant treatment ; Serum treatment ; Aspiration ; its indications ; how to aspirate ; depth of puncture, advantages and disadvantages of aspiration. Injection of quinine and iodine solutions.

The several measures that have been advocated for the treatment of liver abscess are the following :—

Treatment.

- (1) Internal medicines.
- (2) Simple puncture.
- (3) Tapping with aspiration.
- (4) Injection of antiseptic fluids into the cavity.
- (5) The open method of operation—free incision and drainage.

As regards the medicinal treatment of liver abscess, the following drugs have been found useful. They simply allay the effects of concurrent ailments but are incapable of curing the abscess, when once formed.

1. Mercury is not only insufficient but injurious, and should not be given except as an occasional purgative.

Preparations.
of mercury.

2. If liver abscess be complicated with dysentery or diarrhoea, the bowel complaint should be at once cured,

but hepatitis must not be neglected. He should be kept in bed and restricted to milk diet which is of paramount importance in this grave hepatic disorder. A full dose of ipecacuanha twice daily for 2 or 3 days, should be given. Some prefer salines; others give relief by blue pill or calomel sometimes.

3. If hepatitis occurs after the cessation of dysentery, salines should be administered freely.

4. When the acute stage of hepatitis is passed, ammonium chloride in 15 to 20 grains doses thrice daily, often does good. The bowels should be kept open by cascara, eunymine, rhubarb, and alkaline saline purgatives.

5. For the relief of pain: apply poultices, dry and wet cupping over the hepatic area, and leeches around the anus. Flying blisters may be raised repeatedly by liquor epispasticus. Opium is often required to soothe the patient's suffering.

6. If liver abscess occurs without the association of dysentery: purge the patient freely by the sulphates. Massive hot poultice should be applied externally. The patient should be kept, on a low diet, in his bed.

7. Change of climate: Change to a temperate climate often does good but never should be undertaken during the winter. He should always guard himself against chill.

Formerly, bleeding was much in vogue, but blood-letting will not cut short the morbid process in acute hepatitis. Frerichs is of opinion that it should only be ventured in cases of traumatic hepatitis and in robust plethoric persons, when there is great tenderness, with enlargement of the liver and urgent dyspnoea. It is better that it should not be tried at all, though some benefit may follow it.

Leeches: Much benefit may result from the local application of blisters and leeches. Leeches round the anus act directly on the portal circulation. Four to eight leeches, according to the indications should be applied and relaxing purgatives administered, such as magnesium sulphate, mineral waters, &c.

Ice applied to the hepatic region in acute hepatitis is no doubt of great service. Application of an ice-bag induces ischæmia of the organ and relieves pain.

The method of gradual opening of the abscess by the application of caustic, or thermo-cautery applied over the tumour, formerly much in vogue, is now abandoned. Equally so another method,

Obsolete operations.

viz., that of leaving a rigid trocar in the wound. Puncture by trocar was a favourable method with some operators, the canula being left in for several days and then replaced by a drainage tube. Murray in 1839 stated that the Indian Hakims had immemorially resorted to puncture of both liver and spleen for the dispersion of enlargement with induration. Cameron¹ says: "This is a native mode of dispersing hepatic and even splenic enlargement. Acupuncture with a fine needle is a surgical operation said to be much in use amongst the native Chinese and Japanese Hakims". But in simple puncture, pus often refuses to pass through the canula; so now it has been supplanted by puncture with aspiration.

Expectant treatment: In autopsy, abscesses of the liver have been found to have undergone absorption. It was also believed that it was better not to operate, as patients are said to have been cured by the spontaneous discharge of pus into neighbouring organs. The results of operations some 30 years back were far from satisfactory. So it was thought prudent to wait for some fortunate, accidental solution of the difficulty. But now the operation has been simplified,—any medical practitioner, with the aspirator, can operate single-handed and at once; and the results show a decided improvement in the percentage of mortality.

Serum treatment: Rolleston hopes that "probably before long it will be possible to treat this condition (the period of uncertainty) by the hypodermic injection of a bactericidal serum which is polyvalent and able to counteract the microorganism most probably present, as shown by an investigation of agglutinating properties of the patient's blood serum".

Now the surgeons always prefer aspiration when in doubt. It is at once a diagnostic and often a therapeutic measure of great value. Even if pus be not detected, withdrawal of a few ounces of blood from the liver is often followed by amelioration of many distressing symptoms *i.e.*, fever, pain, cough, &c.

The indications of aspiration are the occurrence of repeated rigors and hectic rise of temperature and the persistency of fever and local symptoms. The occurrence of pleural friction in a suspected case of liver abscess is a sign of the extension of the inflammatory process to the thorax and is a signal for operation to prevent its rupture into the pleura or lung.

When a pulmonary abscess consequent on an hepatic abscess is diagnosed, no time should be lost in operating upon it, to prevent further destruction of the substance

of the lung. When we aspirate we must be prepared to open the abscess, if pus be detected, for then we avoid a double operation and a second administration of the anæsthetic. Anæsthesia is absolutely necessary in aspiration, for more than one puncture might be necessary. It is always better to use a full sized aspirator needle so that the thick pus may not block the lumen of the tube.

The site of aspiration. The localising signs are abnormal dulness, a tender area, a constant pain, localised bulging,

localised pneumonic crepitus, and pleuritic
Site.

or peritoneal friction. If on firm pressure of the finger between the ribs pain is complained at one particular spot, it becomes a valuable diagnostic symptom of the presence of pus in the liver. If these be absent the usual site is the upper and back part of the right lobe. The needle may be entered in the right axillary line through the 7th or 8th intercostal space or just below the rib in the right nipple line, or over the centre of the area of dulness behind, below the lung in the line drawn downwards from the angle of the right scapula. The knowledge of anatomical relations of the pleura and the lung in the right side may deter a surgeon from puncturing these structures but the danger is more imaginary than real; and it has been found in practice that punctures of the pleura and the lung are not generally followed by any

untoward result. Moreover an abscess on the dome of the liver may be missed by adhering to the anatomical limits of the safe site for puncture. Try to observe the sub-pleural and sub-pulmonary limits if you can during aspiration but these must not engender the hesitancy to puncture higher, if you are suspicious of the presence of pus there (Manson).

The Modus operandi: The skin should be rendered thoroughly aseptic, and the surgeon should be sure of his instruments as regards their sterilization before puncture. The needle, after making the aspirator ready, is inserted in the axillary line in the 8th, 9th or 10th costal interspace about 1 or 1½ inch from the costal margin and well below the limit of the pleura. It is then carried, inward, upward and slightly backward. If pus be not detected, the needle is to be withdrawn; then transfix the dull hepatic area systematically, both in front and behind, regard being had for the important structures concerned—the lung, pleura, gall-bladder, large vessels and the intestines. But if there is complete absence of breath sounds, of vocal fremitus and resonance over the lower part of right lung and pus has not been reached, then the needle is to be thrust in anywhere below the line of nipple and angle of scapula, disregarding the pleura and lung. Not

more than 6 punctures can be allowed. If pus be not found, wipe the puncture wounds clean, apply a coating of collodion on a film of wool, which should be secured by a few turns of a broad roller. Should, however, pus be found, the needle is left in the liver as a guide to the knife.

The depth of the puncture : "It may be taken as a broad rule that no needle or trocar should be introduced to a greater depth in the liver in the direction of the vertebral

column, horizontally from the surface,
 Depth of puncture. than 4 inches."

Cantlie had his needle of the aspirator and the trocar and canula marked in inches, and the instrument makers should do well to utilise this precaution.

How can we be sure that the end of the needle is inside the abscess cavity in the liver?

"The movements of the needle after it is introduced into the liver aid in the diagnosis of whether or not the point is in an abscess cavity. When the needle is imbedded in the liver tissue, the part of the needle external to the chest or abdominal wall move up and down with the respiration; but should the needle lie in a cavity, a more pronounced swing or pendulum-like movement (Manson) is communicated. * * * * When the pendulum-like movement

is very limited, it may serve to indicate that adhesions have taken place between the liver and its surroundings; but when it is entirely absent, then the needle, even if pus is drawn off is not in the liver at all" (Cantlie).

Sir J. Fayrer is a strenuous advocate of puncture for exploration but insists on opening by incision immediately pus is detected.

As regards the advantages of aspiration, it has been claimed that even if pus be not detected, hepatic phlebotomy is a measure of proved value in hepatitis. The risk of hæmorrhage at a minimum and armed with aseptic precautions, aspiration should be the rule of surgical practice. No ill effects follow this procedure even though blood may leak into the peritoneal cavity. Fagge and Whitla quote instances of the cure of hepatic abscess after simple aspiration and withdrawal of the purulent contents. Easmon¹ also reports such a cure. Cantlie observes: "The abstraction of blood from an inflamed liver is the readiest way of relieving hepatitis". He advises us to withdraw six to ten ounces of blood. Laurie of Hyderabad gives a table showing 4 deaths out of 26

Advantages of
aspiration.

¹ Lancet, August, 13, 1887.

cases of liver abscess which were aspirated antiseptically.¹ Cameron² never had the slightest ill result beyond a little local pain "and he found such puncture followed by gradual absorption and disappearance of the enlargement" Palmer³ says: "In no case do I remember any bad consequences resulting from such punctures." Harley⁴ also advocated the practice. Bérard⁵ mentions a case who was considerably relieved by 4 punctures made into the liver.

But aspiration has its black side too. Many object to Disadvantages. aspiration for the following reasons:—

1. "Extensive suppuration may exist and yet be missed in the aspiration, particularly when the branches of the portal vein are distended with pus." (Osler)

2. Statistical data regarding the actual value or even the safety, of the method of thrusting trocars into the liver in search of pus has not been published. Maclean,⁶ at Hyderabad gave this operation a patient trial but "without a fortunate issue in a single example."

¹ Annual Report on the Civil Medical Department of Hyderabad 1891.

² Lancet, 1863, June 6 and 13.

³ See Fayrer's Work. P. 213.

⁴ See Chapter on Hepatic Phlebotomy. pp. 161 and 509.

⁵ Bérard: Lyon Medical May, 8, 1912.

⁶ Lancet, July, 18, 1863.

3. It should be remembered that an hepatic abscess always begins from multiple foci (Rokitansky) so it might require repeated aspirations.

4. Aspiration of liver is not without danger. Fatal cases are known to have occurred in the practice of eminent surgeons. Many instances of fatal syncope are recorded. Willet¹ mentioned a case in which, aspiration in a doubtful swelling of the liver, killed the patient within 2 minutes. "The sudden, fatal syncope seemed due to the impression made on the nervous system ² through the solar plexus". Hatch ³ and Maitland ⁴ report cases whose death has followed exploratory puncture. Maitland advises us that if a second puncture is necessary, an entirely fresh one should be tried, and not to push the end of the trocar in another direction, as bleeding is more apt to occur there. This is more likely to happen if the patient be the subject of cachexia from splenic leucocythæmia.

5. "Aspiration repeated once or twice, has been frequently employed, but is of little value, and not a few cases are on record in which septic peritonitis or pleurisy has followed the introduction of the needle from the front

¹ B. M. J. November, 13, 1886.

² Jacobson and Rowland. The Operations of Surgery. VIII p. 539.

³ I. M. G. April, 1898.

⁴ B. M. J. 1902, M.L. 458.

or side respectively¹". Sir J. Fayrer remarks that in a case "after death which quickly followed the puncture with an aspirating needle, blood was found extensively extravasated and coagulated in laminae in the peritoneal cavity".

6. We know a case in whom fatal hæmorrhage occurred after aspiration. Lt.-Col. Brown told us of another case, a friend of his, who was aspirated by a noted surgeon of Calcutta and who died two days after the operation from hæmorrhage.

7. And it may be the means of spreading infection in the liver by passing through the abscess and carrying the pus into other parts of organs. Cases are on record where suppuration has followed the aspiration. "As the tenderness involved the left lobe, the abdomen was opened and the liver was palpated and repeatedly aspirated with a negative result. * * * * Four weeks after admission, the fever became higher and on the 38th day after his admission a second operation was performed and an abscess opened in the very position which had been proved to be free from pus by aspiration 29 days before."² Rogers concludes that because ipecacuanha was not tried in this case, abscess was the result; and that other medicines, which were tried in the case, are useless in such

¹ Rose and Carless. Manual of Surgery 4th Ed, p. 908.

² Rogers. Calcutta Medical Journal, 1908, p. 366.

a disease. Might not we argue that aspiration was the cause of suppuration, especially when pus was discovered exactly where its absence was proved before. Suppuration occasionally took place even after taking all precautions against infection from the instruments employed.

8. The trocar may be blocked and the thick pus would not flow through it.

9. "The abscess may leak into the general peritoneal cavity and set up general peritonitis; severe hæmorrhage may occur if the aspirator wounds a large branch of the hepatic artery, or the aspirator may perforate one of the hollow abdominal viscera, such as the stomach, intestines" (Rolleston.)

We have, however, never witnessed any evil effect from aspiration. It used to be frequently done in the S. N. P. Hospital and we have done it many times, but no untoward event happened in our cases.

Observer. Cases of aspiration. Result.

Hammond	8	No bad result.
Bartholow	2	Improved the symptoms.
Condon	11	8—abscess evacuated.
		3—no abscess but improved.
Ramirez		No evil result.
Harlev		Relief.

We may take the advise of Godlee¹ as our guide. "If the existence of an abscess be only suspected and the patient be loosing ground, it is right to puncture the liver in the most likely situations, bearing in mind that, though usually quite harmless, a slight amount of risk accompanies this very trivial operation. If on the other hand, this abscess have ruptured or not, there are no means of diagnosing the whereabouts of the matter, and the patient be not loosing or even gaining ground, the surgeon should hold his hand for a time". Cantlie² considers the inferior vena cava the only vessel likely to be injured and so prohibits introduction of the needle more than $3\frac{1}{2}$ inches. But aspiration is unadvisable if leucocythæmia or anæmia be present and in all cases an examination of the blood from the finger will aid the diagnosis.

Some surgeons prefer, after aspirating the liver pus, to
 Injection of inject into the cavity antiseptic solutions to
 quinine. kill the germs and so to cure the disease.
 Losech found that a solution of quinine 1 : 5000 quickly killed the amœba and so he injected this solution into the bowel of patients suffering from amœbic dysentery. The patients improved and the amœba disappeared from the

¹ Lectures on the Surgical Aspects of Hepatic Abscess. B. M. J. Jan. 1890.

² Cantlie J. B. M. J. 1913, Vol. II. P. 656.

stools, only to re-appear however when the quinine injections were let off. Rogers suggested its trial in the treatment of liver abscess, first drawing off the pus by aspiration, injecting the quinine solution into the cavity and then withdrawing the canula¹. Both favourable and unfavourable reports have been published as regards the treatment; but no definite statistical data are availing. Rogers and Wilson² describe two cases of amœbic abscess of the liver cured by aspiration and injection of quinine into the cavity without drainage. In two cases we tried the treatment, and in both it failed. Some like injection of iodine, but it is often found to be worse than useless. The injection of bichloride of mercury or of iodine solution into the liver is dangerous; as a curative measure it is most uncertain.

¹ B. M. J. September 20th, 1902.

² Rogers L. and Wilson R. (June 16th 1906), "Two cases of Amœbic Abscess of the Liver" B. M. J. Vol. I. p. 1397.

CHAPTER XII.

The Surgical treatment: The open operation, its objections; its merits; conclusions. The methods of operation: I. Abscess struck below the costal border: Abdominal operations: (i) Direct incision and drainage. (ii) Hepatotomy at one operation; with adhesions; (iii) Hepatotomy in 2 stages, when there is no adhesion. II. Trans-thoracic operation: (i) Operation at one sitting; (ii) Operation à deux temps. III. Hepatotomy through a lumbar incision. Zancarol's method. Graves' method. Manson's method, its advantages and disadvantages. MacLeod's method. Fayrer's Method. Rogers' method; Cantlie's observation on the advantages of trocar and canula. Are we to wait for adhesions? Diagnosis of adhesions. Dressings. After treatment Time for healing. Treatment of the Complications. Duration of life after operation. Causes of failures. Cases unfit for operation. Conclusions.

The consensus of opinion of most modern surgeons is for the open method of operation for liver abscess. It consists in early free antiseptic evacuation of the pus by incision.

This method has its opponents and supporters. The main objections raised against the operation are as follows :—

1. That air will be introduced into the abscess cavity and excite fresh inflammation or pyæmia. There is no

doubt of some truth in the observation. The pus at the time of operation is often very viscid and thick, often it can not pass easily through the tube; but after a few days, the pus becomes thinner, and less viscid, and will be found on examination to contain many streptococci and staphylococci, which are no doubt due to secondary infection. But the same result will follow if the abscess opens spontaneously into the bowel, lung or externally.

2. That pus may escape into the peritoneum and excite fatal peritonitis. Generally we find adhesions already formed in abscess of the liver, as a safeguard against the accident. Morehead speaks of absence of adhesions as quite exceptional—in only 3 out of 76 fatal cases. Moreover, we can produce adhesions if we like them before opening the abscess. Even if pus finds entrance into the peritoneal cavity, the risk and danger is small if free drainage to the outside is secured.

3. That the mechanical injury is apt to produce hæmorrhage and fresh hepatitis. This objection is found on theoretical grounds, and we have never met with such an accident in the hospital.

4. That gangrene may supervene and kill the patient¹. This also occurs in a spontaneous rupture. The gangrene

¹ Maclean. *Lancet*, July 18, 1863. Budd. *Diseases of Liver*, p. 124. Cruvelhier. *Anat. Path. Liv.* 40.

is often connected with the caries or necrosis of the ribs, which may be averted by opening the abscess early. This is said to be "one of the deadliest accident to which the victim of liver abscess is liable" and no time should be lost in opening the abscess.

5. Budd and Low ¹ think it better to allow an abscess of liver pointing to the abdominal wall to open of itself for the following reasons, besides those mentioned above :—

i. Because the inelastic structure of the lobular substance of the liver does not permit the cavity to contract when a free opening has evacuated the pus.

ii. When the operation is left to nature, small worm-eaten-like openings serve to discharge the pus, so that it has slow but constant escape. As these apertures never close up, and as the matter is always oozing out, air can not enter, no septicæmia occurs and no secondary fever sets in. But the case reported by Budd in support of this hypothesis really speaks against him as it took 4 years for the abscess to heal up. The vital power of the patient at this stage is usually not "adequate to the long process of ulceration through the textures" (Cooper ²). By proper antiseptic precaution we may avert the danger to a certain extent. Again the same difficulties would occur in spontaneous

¹ Madras Quarterly Journal, April, 1863.

² B. M. J. May 23, 1863.

rupture externally. External rupture though one of the most favourable modes of termination, is rare indeed.

Frerichs ¹ and Morehead ² advocate opening the abscess in selected cases; while, others like Murray, Cameron ³ and Martin ⁴ would not lose a day, if they be sure of pus formation. They urge that by non-interference the dangers would be the following:—

1. The abscess daily becomes larger and larger and ultimately the whole of the hepatic structures may be destroyed, and necrosis of the ribs results.

2. That sudden death is possible from the abscess bursting into the pericardium, peritoneum, or the pleura.

3. The majority of the patients often die of exhaustion from hectic fever or diarrhœa, either while the abscess is still confined to the liver or after it has ruptured. Fayrer says: "Cases have not unfrequently occurred where simple incision, made earlier, would in all probability have relieved suffering, and might have saved life".

The statistics of operation clearly show that by early operations, we can often reduce the mortality. We have achieved such encouraging results by operation, that we do not like to lose any time by the expectant mode of

¹ Disease of Liver. Syd. Soc. Ed. II, p. 147.

² Researches on Disease in India, 1860, p. 410.

³ Lancet, June, 6 and 13, August 8, 1863.

⁴ Lancet, August 20 and 27, 1864.

treatment. The result will compare favourably with the statistics of previous surgeons.

Observer.	Cases operated.	Result.	Per cent.	Mode of operation.
Curtis ¹ .	{ 10 in 1772 14 in 1773	{ 2 4 } recovered	25.	
Waring.	81	66 died or	81.4	Trocar or knife.
Morehead.	24	16 „	66.6	
McConnell.	14	6 „	„	Aspiration.
Condon.	8	4 „	„	„
Sachs.	21	13 „	„	„
De Castro.	22 large. 10 small.	Recovered „	31.8 70	„ „
Fletcher.	17	12 died or	70.4	„
Strong.	12	9 „	66.6	„
S. N. P. Hospital				
1905.	9	2 „	22.2	Open method.
1906.	6	1 „	16.6	„
1907.	15		13.3	

In the S. N. P. Hospital we always assisted the surgeon when operating these cases; they generally had single abscess of the liver; and we always made a careful selection of our cases, and observed the strict rule of antiseptic precautions. In all these cases we first aspirated and being sure of pus, opened the abscess. This unusually happy result may seem extraordinary. But we have found that the natives suffering from abscess of the liver are better subjects for operation

¹ Goodeve. Sketch of the progress of European medicine in the East.

than the European. And we are supported in this inference by the observations of Jackson¹ who says: "In the natives of India there is much greater proportion of superficial abscesses that point towards the abdominal muscles; and as in them the febrile action is much less severe, the abscesses are more limited in extent, and can therefore be more beneficially treated by artificial means than is the case with the Europeans. In 9 cases of the disease in natives, under my care in the year 1853, in which there was operation by puncture through the abdominal muscles, 8 were successful. In Europeans a successful issue of abscess of the liver consequent on operation is extremely rare, and my experience with respect to them disposes me to object, most generally, to operation in their case". Moreover of 203 cases collected by Rouis, where the abscess was not opened, 162 or 80 percent. died. We may take however as our guide the conclusions arrived at by Murchison:—

1. In all cases when there is a visible fluctuating tumour, operate at once.

2. In cases where the symptoms of abscess of the liver accompany a distinct tumour, it is well to operate.

3. If symptoms of abscess co-exist with enlargement of liver but no tumour, if there be any local œdema or

¹ Dr. J. Jackson. Notes on the management of the Liver Abscess. *Lancet*, Aug: 1859.

obliteration of intercostal space, or localising pain, it will be well to operate (Frerichs).

4. If local signs be absent, but constitutional symptoms be decisive, aspiration is advisable.

5. When multiple abscesses are suspected, it is better not to operate.

6. We would add that if it be impossible to diagnose the seat of the abscess or when the patient is evidently sinking, let him die in peace. We have often seen moribund cases to be operated in hospital. It does no good to the patient, nor enriches the fame of the surgeon.

The operation usually practised by the English surgeons has been described by Godlee.¹ He and other surgeons contend that if pus has been discovered, "the best plan is to give exit to it at once making a hole large enough to admit the finger, and afterwards a drainage tube of the same size". But there are reasons against such a practice though it is advocated by Manson and Cantlie:—

1. The canula and tube may slip out.
2. The drainage is inefficient.

¹ B. M. J. January 11, 1890. See also Debate at Brit. med. Asso. Meeting at Brighton 1886 and An. of Surg. Oct. 1886 and March 1887.

3. If the pus leaks into the peritoneal sac, it does so unseen.
4. The trocar may puncture important parts.
5. Puncture and drainage would be useless, where more than one abscess existed.

Now we shall describe the various methods of operation. The liver may be approached either through the abdominal or thoracic walls. It is best to approach an hepatic abscess through the abdominal wall when practicable.

I. Abscess struck below the costal border: Abdominal operations:

(i) *Direct incision and drainage:* It is indicated when the abscess is "pointing," when tenderness, œdema and redness make it probable that adhesions exist. A free incision 4 or 5 inches long is made and the parietal peritoneum united to the subcutaneous tissue of the wound. A fine trocar or aspirator needle is thrust in to verify pus formation, and tampons of iodoform gauze are carefully packed around. The abscess is then incised, the opening plugged, and freely dilated with the finger. Any escape of pus into the peritoneal cavity is prevented (1) by the careful packing; (2) by the finger hooking up the liver against the wound; (3) by an assistant keeping the parietes steadily against the liver. All bleeding is arrested by forceps or

sponge pressure. If the abscess does not empty itself readily, a large tube may be pinched and placed at the bottom of the abscess, when it will act as a syphon (Greig Smith). Gently explore the cavity with the finger, and if a second abscess be found, open it with the finger. The opening is now plugged, the liver and the parietes are pressed together, the tampons are removed, and the edges of the hepatic incision are stitched to those of the incision in the abdomen with silk sutures passed with curved needles on a holder. A large rubber tube is passed into the abscess cavity and fixed to the edges of the parietal wound by a salmon-gut suture. A thick dry gauze dressing should be frequently renewed; and it is advised to secure it by a many-tailed bandage.

As regards the danger of the routine use of the anæsthetic in cases of operation for liver abscess, or at least such administration combined with the usual preliminary starvation, reference may be made to a paper by Stiles and Macdonald, which gives the bibliography, and to an article by Hunter who states that the delayed chloroform poisoning dependent on the state of the liver may be prevented if instead of withholding food, the patient be given a very nutritious and easily digestible meal, well sweetened, 2 or 3 hours before the operation.

Dangers of
Chloroform.

(ii) *Incision and drainage by abdominal section at one sitting: Hepatotomy at one operation.*

Indications: It is advised when we are sure that adhesions are already formed; the aspirator needle is kept as a guide.

Position: The patient is placed in the dorsal position, or he lies somewhat on the sound side, if incision is to be made on the lateral aspect of the body. A sand-bag or pillow is placed underneath the lower dorsal region of the spine so as to lift the liver forward nearer the parietal wound. The surgeon stands on the right and the assistant on the left of the patient.

The operation of hepatotomy may be divided into four stages:

(a) *Parietal incision.*—The skin is well cleaned over the most prominent part of the swelling. An abdominal incision 3 inches long is usually commenced at the costal margin and deepened down to the peritoneum. Some recommend the incision to be one inch and a half in length at first, and it is to be extended after the preliminary digital examination has been made into the abdominal cavity.

(b) *Exposure of liver, incision of wall of abscess, and evacuation of pus.*—A pair of sinus forceps is pushed along the needle through the hepatic tissue into the abscess

cavity. The aspirator canula is then removed ; the blades of the forceps are then opened and withdrawn. Insert the fore-finger and move it round to enlarge the wound and to ascertain the size and direction of the cavity. If there is no great thickness of hepatic tissue between the surface and pus, a knife may be employed for the purpose of opening ; an incision $1\frac{1}{2}$ inches long is made in the hepatic substance over the abscess. All hæmorrhage is arrested from any hepatic vessel by ligature. But if the abscess lies deeply, it has been suggested that the thermo-cautery should be employed so as to prevent bleeding by sealing the mouth of the hepatic veins.

(c) See page 242.

(d) *Establishment of drainage.*—When the abscess is not large, the interior should be packed with a long strip of antiseptic gauze, one end of which protrudes from the external wound. This should be changed daily. Some prefer a flanged rubber drainage tube, about the size of the finger and 3 or 4 inches long, which is introduced by a pair of forceps to the back of the abscess. Pus flows out, but when it no longer flows freely, a gentle examination of its interior may be made, to ascertain if another abscess exists. The abscess cavity should be cautiously cleaned out by means of a soft sponge on a holder, or the cavity may be irrigated with an antiseptic solution, and the aperture temporarily

plugged with a sponge on a strip of gauze. A massive antiseptic dressing is then applied and secured by a binder or better by a many-tailed bandage. The tube should not be taken out till the discharge has almost stopped, but it should be made shorter if the length be inconvenient. Numerous successful cases have been reported by Lawson Tait, Thornton, Bryant and other surgeons.

There are objections to the use of a drainage tube, at least immediately after the operation :

1. It allows venous oozing to take place from the abscess wall.
2. If the tube be too long or too stiff, the lower end may lacerate the wall of the abscess cavity during respiration.
3. The aid of capillary attraction is wanting in the case of a drainage tube.
4. If the tube be passed too far in, it may cause sloughing from pressure upon the abscess wall which may be thinly separated from peritoneum in places.

(iii) *Incision and drainage by abdominal section in two stages: Hepatotomy in two stages (à deux temps): Volkmann's operation :*

Indication : If adhesions be not formed or be weak.

¹ B. M. J. Nov. 13th 1886.

Incision: An incision, 4 inches long, is made over the abdominal swelling. The bleeding is stopped and peritoneum is slit open.

(c) *Union of margins of hepatic incision to those of the parietal wound.*—A double circle of stitches is passed through the liver capsule and the abdominal wall.¹ The outer row should be interrupted sutures, the inner continuous. Some surgeons recommend a single ring of interrupted silk sutures, uniting the margins of the hepatic incision to those of the incision in the abdominal wall. The sutures are advised to be one-quarter of an inch apart; and each suture is entered at the deep part of the hepatic incision, and it emerges about $\frac{1}{2}$ inch from the margin; it is then passed through the parietal peritoneum and the fascia and muscular tissues of the abdominal wall.

Beyond the upper and lower ends of the hepatic incision, a suture should be passed through the abdominal parietes and hepatic substance and finally through the abdominal wall on the other side of the incision. To make certain of the position of pus, a fine trocar may be thrust in. Then the sinus forceps is pushed in as before. After stitching, some prefer, before opening the abscess, to stuff the wound in the abdominal wall with aseptic gauze and to wait for a day or two for the formation of adhesions and then

¹ Barwell, *Lancet*, January, 1887.

to open it as before. It is often advised to wait longer, 3, 4 or 7 days. Some do not stitch at all but merely stuff the wound with gauze (Graves), and endeavour by well-adjusted bandaging to keep the abdominal parietes in contact with the liver. This is the method recommended by Bégín and Recamier who wait for 3 days after opening the peritoneum and then the pus is let out.

The methods of treating an hepatic abscess by abdominal section have the following advantages :—

1. The benefit of a free incision and thorough drainage.
2. The surgeon can see what structures he is dealing with.
3. Bleeding from the liver can be seen and arrested.
4. Pus can be prevented from escaping into the peritoneal sac by packing, &c.

Comparison of the two methods. The direct method by one stage is the more satisfactory. The precautions necessary in the method by two stages are not well founded. The risk attending the escape of pus into the peritoneal sac has been much exaggerated. In the two stage method much time is lost, which is of paramount importance in the case of an abscess of the liver.

It is suggested that the incision on the abscess may be made without giving a second anæsthetic. But such

practice is alarming and painful to the patient; and the evacuation of the abscess cavity is better done under chloroform.

II. The abscess is struck through an intercostal space: Transthoracic operation.

Indication: This operation is requisite in the following clinical conditions:—

(a) Subphrenic abscess which is not accessible through an abdominal incision.

(b) Abscess of the superior part of the liver which extends upwards into the thorax.

(c) Subphrenic hydatid cyst.

(d) When the abdominal operation gives insufficient drainage.

The transthoracic operation may be done in one or two stages as in the case of operation through the abdominal wall.

(i) Operation at one sitting:

Position:—The patient lies somewhat on the left side. The surgeon stands on the side of the patient, and the assistant on the opposite side.

Incision: If the abscess be pointing in the side, in order to avoid the pleura the incision should not

be more than 2 inches above the margin of the ribs in the mid-axillary line. It may be remembered that the lower reflection of the pleura forms an oblique line running a little below the 6th rib in the nipple line, the 8th rib in the axillary, the 10th in the parascapular and the 11th at the spine. The incision, 3 inches long, which may be extended if necessary, is made parallel to a rib.

Excision of the rib: If space be not sufficient for manipulation and drainage, Kartulis recommends excision of 2 inches of the rib. The rib is exposed, the periosteum and structures which are attached to it are separated and then a portion of it is excised, the section probably passing through the cartilage. This can easily be done by means of the rib-shears. If there be adhesions, two courses are open to the surgeon: (a) The diaphragm may then be stitched to the thoracic wall and skin, and the abscess is opened as before. To stitch the the capsule of the liver with diaphragm is difficult, and Macleod thinks, stitches would not hold in a soft and inflamed liver tissue, but it should be tried, if there be no adhesions formed. This is specially to be observed if the liver be opened at a spot where it is covered by the peritoneum. (b) Otherwise the general serous cavity must be carefully protected by pledgets of gauze or sponge, before letting out the

pus, and the assistant must keep the parietes in close contact with the hepatic tissue. If the underlying costal layer of pleura be divided and the cavity of the pleura be opened, the air rushes inwards and the lung of the corresponding side becomes collapsed. The diaphragmatic layer of pleura is next incised. It will be necessary to stitch the diaphragm to the costal pleura, otherwise pneumo-thorax would be the result, an unpleasant but not a serious complication. The margins of the wound in the costal and diaphragmatic layers are closely united with a continuous silk suture. It may be necessary to aspirate the liver swelling so as to relax the diaphragm and the pleura. Godlee advises us to stitch the hole in the pleura before dividing the diaphragm to open the abscess; this is necessary as by the aspirating influence of inspiration, pus may enter the pleural cavity.

Some even ignore the peritoneal adhesion and do not stitch the peritoneal surface before opening the abscess. We often omitted stitches and did not get any bad result. The danger of introduction of pus into the peritoneal cavity is small if there be good drainage externally. It is often very difficult to apply these stitches, as the hepatic tissue becomes very friable. Macleod even thinks that the removal of a portion of rib is unnecessary. He uses metal drainage tube to prevent nipping of the

¹ B. M. J. Oct. 22, 1887.

rubber tubes and for facility of introduction. These tubes are 4, $3\frac{3}{4}$, or $2\frac{1}{2}$ inches long and have an oval lumen, the diameter being $\frac{4}{10}$ or $\frac{3}{10}$ th of an inch long. A special guide is used for introduction of the tubes.

(ii) Operation à deux temps: The early stages are similar to those described before. After suturing the costal and the diaphragmatic layers, the wound is plugged with aseptic gauze, and left for 3 to 5 days. Then the liver is incised and treated as before. In this operation the parts become coated with lymph, and the anatomical structures cannot be recognised.

III. Hepatotomy through a lumbar incision: Occasionally abscess of the liver presents in the lumbar region, then the surgeon should select this route of attack, since the abscess can be opened without incising the peritoneal sac, and very good drainage established.

Incision: Incision is made in the lumbar region of the right side, parallel with the lower margin of the last rib, and it is deepened through the fascial and muscular strata to the posterior aspect of the liver.

The liver is now incised, the cavity of the abscess is reached, and treated as has been already described in the abdominal and transthoracic operations.

The extremities of the incision in the abdominal parietes may be closed with sutures, if desired and the cavity drained by a tube and gauze packing.

Sometimes the abscess is at the back and can only be operated by a posterior incision. The pleura must be opened, but the pleural aperture should be closed before opening the abscess. Godlee advises us to make "a rather free transverse incision into the diaphragm by passing a strong catgut continuous stitch right through its substance and through the structures occupying the intercostal space; viz. the intercostal muscles and pleura. Those who possess the means of operating with the "*Oberdruck* or *Underdruck*" apparatus would no doubt find the operation much simplified by the avoidance of pneumothorax."

Zancarol and other continental surgeons advise that Zancarol's the hepatic and integumental incisions method. should extend the whole breadth of the abscess cavity. He describes a method of completely cleansing the cavity by irrigation, which he employed in 50 cases with success. Some French surgeons recommend scraping the abscess cavity and most practise irrigation with some antiseptic fluid,—measures which we have never adopted in our cases. It should be noted that

the pus in the tropical liver abscess is practically free from bacteria.

Some surgeons use a paquelin knife with the view of minimising bleeding. We have never met with any serious bleeding from the incisions and it would be better not to use the knife deeply into the liver, where finger should always be used.

According to Zancarol, the mortality after operation depends on the method used by the surgeon :

<i>Treatment.</i>		<i>Mortality,</i> <i>per cent.</i>
Puncture with a trocar or aspirator needle ...	80	
Incision with knife ...	71	
Thermo-cautery ...	50	
Non-curable ...	40	
Curable ...	60	
Cured ...	77	
Fatal ...	71	
Result unknown ...	9	
Total cases ...		157

Graves¹ of the Meath Hospital suggested another mode of operation. It is based on the Grave's method. fact that in several cases, an incision over a deep seated abscess often fails to give vent to the pus in the first instance, yet in the course of a few days the pus finds its way to the incision, and bursts through it. He therefore made an incision 4 inches long over the tumour and then carried it deeply to within about one or two inches of the peritoneum. It was then plugged and left open; after 2 or 3 days the pus would find its way through the incision. But often we can not delay so long when operation is decided. Neither can we be sure that the pus will take this route and will not escape into the lungs or bowels. Budd tried this method with unsatisfactory results².

Manson describes a method of operating on the abscess . Manson's of the liver. The apparatus consists of method.

a large trocar and canula, a steel stillete, two metal buttons and a piece of drainage tubing. The ends of the tube are stretched and then lashed to the stem of the buttons, over the shorter end of which it is also tied. The tube is then mounted and stretched on the stillete by inserting it through one of the drainage holes.

¹ Dublin Hospital Reports Vol, IV P. 40.

² Disease of the Liver P. 123.

The tube then can easily pass through the canula. The aspirator needle is withdrawn and an incision one inch long is made with a scalpel. The trocar and canula are then thrust into the abscess and the trocar is withdrawn. Then the stretched tube is slipped into the canula and carried to the back of the abscess. Holding the tube well, the canula is withdrawn. The button on the free end of the apparatus is slipped off the stillete, the end of which is made to perforate the drainage tube. When the tube has completely contracted, the stillete is withdrawn. The tube is then transfixed with a safety pin and superfluous tubing cut off. Pus issues freely and the tube plugs the wound in the parietes and the liver, and bridges the peritoneal cavity. In this way, extravasation of pus into the pleura or peritoneum is effectually avoided. Cantlie introduces a few modifications in the instrument and he prefers "a drainage tube open at the end and stretched merely upon a metal rod with a blunt hook at one side so as to catch and stretch the tube".

He claims many advantages for this operation :

Its advantages
and disadvantages.

1. Easily done. 2. No assistants necessary.
3. No risk of bleeding. 4. Pus can not escape into peritoneal cavity. 5. A larger drainage tube may be substituted. 6. Deep abscesses may be opened easily. 7. Less shock. 8. No risk of pneumo-

therox even if pleura be opened. 9. Good drainage. The device is no doubt simple and ingenious. But we can not recommend it for the following reasons :

1. Inefficient drainage :—in 3 cases it was tried but the cavity could not be properly drained. The pus is tenacious, but Cantlie observes: "The syphon drainage obviates this and promotes a continuous flow".

2. The tube may be nipped by being tightly grasped. It may be displaced and then difficult to reintroduce it. When tube gets out, pus may enter the peritoneal cavity easily. To obviate these defects MacLeod uses metal drainage tubes.

3. The tube can only be of moderate size. It is difficult to know the position, direction and depth of the abscess by the aspirator. It is impossible to explore and break down the septa in the cavity. No instrument can be compared with the surgeon's fingers.

4. In India, it is difficult to keep the rubber tubes in good condition.

5. McConnel says: "We use no special probes, canulæ, apparatus of any kind nor find the need of them. An abscess of the liver is treated like an abscess of any other part of body on general surgical principles, the most important of which are free drainage and anti-septicism".

MacLeod¹ of Shanghai has introduced a trocar and canula which can be inserted into the cavity upon a guiding rod, by which means failure to reach the pus is avoided, its presence being made certain by the aspirator. So Sir, J. Fayrer advises us to use a large grooved trocar and canula devised by himself in cases of deep-seated abscess to avoid hæmorrhage. But he admits that as the cavity contracts, the tube is apt to get twisted upwards or downwards, displaced, even extruded, when it may be very difficult to replace it. In such cases should there be any lodgement of pus, the opening must be enlarged, and it is absolutely necessary that the tube should be re-introduced. It may even possibly be required to make another opening².

Rogers of Calcutta has devised an apparatus for evacuating pus. It consists of a big trocar and canula which acts like Manson's apparatus. We have never seen it used. But we think, after the satisfactory results obtained by open incision, as shown in our statistics, we can not change the open method of treatment for any other method, in which pus is apt to accumulate.

¹ Journal of Tropical Medicine, November 15th, 1900.

² Davidson's Diseases of Warm Climates P. 664-65.

It would be well here to discuss the reasons put forward by Cantlie in favour of trocar and canula and against the operation by incision:—

(1) That the operation is one of much less severity : The severity of an operation can only be judged by the results. An operation may be simple, but if it fails in attaining its objects, it is worse than useless.

(2) Pus rarely escapes into the peritoneal or pleural cavities after puncture by trocar and canula : No doubt this is an advantage but it has been found by experience, that if there be good drainage externally the danger of introduction of pus into the peritoneal cavity may be neglected.

(3) Suturing of the liver to the abdominal wall is impossible : These stitches may be ignored and no harm would result.

(4) Suturing the pleural layers and the diaphragm etc. is usually only a surgical farce, as it is not possible to do it thoroughly : In fact it is not absolutely necessary to do so.

(5) After laparotomy, the liver is exposed but the surgeon cannot localise a deep-seated abscess. He shall

have to use a searching needle and blood and pus may be introduced into the peritoneal cavity: This is an argument against a practice which does not exist. Surely many surgeons do not declare it as "unsurgical and unwise to use even the needle of an aspirator whereby to explore for pus in the liver as a preliminary" step. First aspirate, and if pus is present, operate by incision.

(6) The operation can be done single-handed and outside the hospitals: If trained assistants are not available and if the patient can not be safely conveyed to the nearest hospital, the surgeon is undoubtedly free to choose this operation in an emergency. But we cannot concur with the verdict of the Section of Tropical Diseases at Ipswich: "The operation by incision is unnecessary in its severity and is attended by less favourable result than operation by trocar and canula.

There is no doubt that the trocar is a very useful instrument for the puncture and evacuation of the abscess when it is tending to point on the surface of the body. It is a most valuable instrument for the location and partial evacuation of an abscess, after the liver has been exposed by an incision, and it is to this class of cases that the use of the trocar should be restricted.

Cantlie¹ says :—" When the pus is in the left half of the liver—a rather rare occurrence—do not attempt to confirm the diagnosis by introducing an exploratory needle, nor use the trocar or canula, but cut down upon the liver through the abdominal wall and evacuate the pus in the usual way."

The question "Is it necessary to wait for adhesions to be formed before operation?" requires
 Are we to wait for adhesions ? a few words of explanation.

Surely we must not wait for adhesion. We have always found adhesions well formed in cases who came for operation. Sir Henry Cooper² is of opinion that in all cases in which the suppuration has extended so near the surface of the liver as to give the sense of fluctuation, irritation and adhesion of the surface have taken place. Therefore, he advocates a direct opening into the abscess, when there is reasonable ground for believing that an external outlet for the matter is the direction taken by the abscess. Peritoneal connections are sure then to have taken place. Tenderness of the tumour is the most satisfactory indication for operative procedures, and a hardened mass of effused lymph a certain confirmation of the formation of adhesion. Delay exposes the patient to the risk of

¹ Cantlie, "One hundred cases of Liver abscesses" B. M. J. Vol. II, P. 1342. November 9th 1907.

² B. M. J. May 23, 1863

rupture by coughing, sneezing, etc., and so to laceration of any adhesion already formed. Godlee however demonstrates that even when an abscess is almost pointing, it is impossible to be certain that the liver will be found to be adherent.

To ascertain whether adhesions are formed or not, Budd¹ says: "We may be sure of it by feeling the edge of the liver or some prominent part of its surface, and marking the place of this with a pin on the surface of the belly. If the liver be adherent to the abdominal parietes, the line or spot so marked will correspond to the edge or prominence of the liver in all positions of the body. If it be not adherent, the liver will slide along the wall of the belly when the patient draws a deep breath, or changes his posture—the liver will fall, for example towards the left side when he turns from his back over to that side, and the line or spot will no longer correspond to the edge or prominence in question".

The abscess cavity is generally not disturbed by any dressings. dressings, but others recommend that the abscess cavity should be mopped out and stuffed with iodoform gauze. But the pus is often too viscid to be absorbed by these dressings; it tends to escape or lie between the skin and dressing as a glairy, sticky mass.

¹ Budd's Diseases of the Liver P. 122.

So the dressings should be changed frequently for the first few days. The vicinity of the wound should be well washed with carbolic acid (1 to 60) or bichloride of mercury (1 to 2000) solution. The dressings must be antiseptic; carbolic gauze, or Lister's cyanide of mercury gauze may be used. They must be extensive and if the discharge be copious they will require change in a few hours. We often used carbolised tow over the deep dressings. When the abscess is deep-seated and much liver tissue is to be incised, free hæmorrhage is likely to occur. The wound then must be plugged by the finger for a minute or two, and if this does not arrest it, a plug of antiseptic material must be left in the wound for a day or two.

The treatment of liver abscess after operation should
After treatment. be as follows :—

(1) For the first few days, dressings should be changed frequently as the discharge is considerable.

(2) As the discharge diminishes in favourable cases, the dressing should be changed every second or third day.

(3) The drainage tube should be kept as it is, for once removed it is often difficult to replace it.

(4) Later, remove and clean the tube, and if discharge be less and if it be pushed out, shorten it.

(5) If still the temperature rises indicating retention of pus, rectify the drainage tube. Dilate the sinus with

forceps and finger; and introduce a full sized drainage tube. If the temperature continues to rise, make a counter opening if possible. But in no case should the tube be allowed to remain for a period more than necessary. But Godlee advises us against rapid shortening and removal of the tube for the original abscess cavity may be refilled and so may simulate a second abscess.

(6) If it be septic, flush out the cavity daily or twice a day with a non-mercurial antiseptic fluid. But it is found that cases do better without washing out, even by antiseptic solution. For irrigation, a double or two-way rubber tube is the most suitable.

(7) If still the temperature rises, a second abscess is possible or there is some complication. If a second abscess be the cause, aspirate, open and drain it.

(8) Regulate his diet and attend to the state of the bowels. Maintain the patient's general condition by the administration of plenty of easily digested and nutritious food, with stimulants if necessary. Use chloral or morphia if he be restless or sleepless; and simple tonic with quinine if fever persists. When convalescence is established, the functions of the liver remain torpid and its substance often indurated; in such a state Martin's nitro-muriatic acid bath may be of use. They promote the

depurative functions of the liver, kidneys, bowels and skin.

The time required for healing after operation varies within wide limits. MacLeod mentions that a period of 12 to 15 days was about the time required for an abscess antiseptically treated to heal up. We have found however that a month's time is required for thorough healing of the wound. But when a sinus occurs it may take longer time. "Morel recorded one case, where a seropurulent fluid continued to flow from the wound in the right side, three years after the opening of the abscess. Cassimir Broussais has given the details of another case, where the cicatrix in the epigastrium had to be punctured almost every 2 months." (Frerichs).

Treatment of abscess rupturing into a serous cavity :—

Treatment of the complications.	When the rupture occurs into pleura, or peritoneum, the serous cavity should at once be opened and drained; otherwise death would be inevitable.
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(i) The hepatic abscess may communicate with the right pleural cavity. Make a very free opening into the chest by resecting portions of one or more ribs, evacuate the pus within the liver and drain both the pleural space and the abscess cavity. If there be no communication between the abscess and the pleural cavity, and if there be at the same time an empyema of the right pleural cavity, we

must open, evacuate and drain the two pus-containing spaces through separate incisions. The empyema is treated by incision and resection of one or more ribs.

(ii) If the abscess communicates with the pericardium no operation is necessary as the complication always proved fatal.

(iii) When the abscess has burst into the peritoneum, either a localised encysted abscess may develop by the formation of adhesions or a general acute septic peritonitis results. In the first condition, the abdominal wall should be incised, and the abscess evacuated and drained. In the second class of cases, surgical interference cannot prevent the fatal result. The peritoneal cavity may, however be opened, drained and washed with antiseptic solution.

(iv) Treatment of abscess discharging into the lung :—
If the lung be the route of pus and if still the progress be unfavourable, and the contents are evacuated by the mouth we must interfere if (1) there be continued discharge of pus and blood with hectic fever, or (2) pneumonia or abscess in the lung results. It is difficult to decide whether the abscess should be opened, especially as rupture into the lung is a favourable mode of termination. If the temperature and quantity of expectoration increase, and the patient loses weight, and suffers from a harassing cough, operation should be decided ; if not, we may wait.

In exploring the liver in such a case, as the cavity is often collapsed, the needle should be pushed well in and a good vacuum maintained in the aspirator, and then slowly withdrawn. Keep the needle as a guide in operation and drain the abscess through an incision in the chest-wall. Recovery is possible when the drainage tube is pushed in the presumed direction even though the abscess cavity be not entered and drained.

(v) The hepatic abscess may burst into the alimentary canal as duodenum, stomach, &c., and pus is discharged into their lumen. In such cases, septic material passes from the alimentary canal into the abscess and keeps up the inflammation. Then the overlying portion of the abdominal parietes should be incised and the abscess evacuated and drained through it.

The duration of life after operation varies. In Duration of life after operation. Waring's 47 cases we find

Died the same day	1
Died the following day...	...	7
Died from 2nd to 5th day	...	10
" " 6th " 10th "	...	9
" " 11th " 15th "	...	6
" " 16th " 20th "	...	2
" " 21th " 30th "	...	4
" " 31th " 40th "	...	1
" " 41th " 50th "	...	3
51st day and upwards	4

The total number of days of life after operation in these 47 cases is 842, giving an average of about 18 days to each case. We have also found that the patients after operation often die at the beginning of the third week.

Failure to cure the disease after operation may be
Causes of failure. due to the following causes:—

1. There may be a second abscess which cannot be detected.

2. There may be no tendency to healing in some cases, owing probably to ill-health.

3. After operation, pus may enter the peritoneal cavity. The big liver is generally pushed down and kept in contact to the peritoneal wall, and so peritonitis is seldom developed unless there be a large collection of pus.

4. Inter-current diarrhoea or dysentery. In the S. N. P. Hospital, we often noticed that apparently hopeful cases died of dysentery developed after the second week of operation.

5. Fatal hæmorrhage. Bleeding sometimes gives relief and the patient recovers. Hæmorrhage may be in the peritoneal cavity, but generally it causes no harm unless the bleeding be excessive.

6. Operation of cases unfit for it. Proper selection of cases is of the utmost importance for the success of the operation. Often the patients are operated in hospitals to swell the number of operations performed.

The two conditions excluding the possibility of Cases unfit for operation. success after operation are :—

1. The existence of a plurality of abscess.
2. Dysenteric state of the large intestine.

Thus in Waring's cases we find :

Total Number	300
Multiple abscesses	108
			192

Of the 192 cases in which the abscess
was solitary, there was more or less
dysentery in

... 76

116

“Thus it appears, that out of the whole number, only in 116, or little more than 1 in 3, could the operation have been undertaken with any reasonable probability of success; and this number would, of course be still further diminished, by taking into consideration, the cases in which the abscess, though solitary, communicated with the lung, colon, or some other viscera, and those in which organic diseases existed”.

There is often more than one abscess. This was also the case in 13 out of 29 cases recorded by Annesley, and in a still larger proportion in the cases collected by Andral, Louis, and Budd. Thus the number of abscess is an important factor from a surgical stand-point.

McConnel¹ observes: "Generally a native will try, a hundred remedies, and procrastinate from week to week until he becomes so weak and exhausted that, when he does seek admission, his state is most unfavourable for operative interference. He has no vitality, the abscess has assumed enormous proportions, or secondary suppurative foci have already formed in other parts of the liver, and such cases die in the proportion of quite 50 per cent., probably more. But when admission is sought fairly early in the disease, and the general vitality is not greatly impaired, we have, I should say, a success of quite 75 per cent., by incision and free drainage, with antiseptic precautions. And the same holds good as regards private patients, consultation here being early, and the chances of recovery greatly enhanced."

Conclusions :—Thus we have tried to show that:

1. There are many difficulties in accepting dysentery, though commonly held to be so, as the sole cause of liver abscess in the Indians.

¹ Davidson's Diseases of Warm Climates, P. 684.

2. Perhaps it depends upon a combination of causes : Climate, Dysentery, Malaria and Alcoholism.

3. Many of the hepatic symptoms described in the text books as typical are not found in the Indians.

4. The value of leucocytosis in diagnosing the pre-abscess stage is questionable ; and so of the ipecacuanha treatment in its cure.

5. The natives are better subjects of operation in this disease than Europeans.

6. The open method of operation under antiseptic precaution is the best treatment of liver abscess.

(a) If the formation of pus could be ascertained, hepatotomy at one stage is the best surgical procedure.

(b) A large drainage tube should be used to ensure free exit of pus.

(c) Use antiseptic dressing but need not wash the cavity as a rule.

In the preparation of this subject full and free use has been made of the works mentioned in the Bibliography.

THE END.

Appendix I.

STATISTICS OF CASES.

No.	Name.	Age.	Caste.	Cause.	Treatment.	Result.
1	P. N. D.	42	H. M.	Alcohol	Opened (late)	D
2	P. D. B.	38	H. M.	"	"	R
3	D. N. H.	36	H. M.	"	"	R
4	K. C. B.	45	H. M.	"	"	R
5	M. N. M.	49	H. M.	"	"	R
6	H. D. R.	32	H. M.	"	"	R
7	H. N. B.	39	H. M.	"	"	D
8	R. M. S.	28	H. M.	Gooly	Burst Externally	D
9	S. C. B.	42	H. M.	Alcohol	Burst into Lung	R

No.	Name.	Age.	Caste.	Cause.	Treatment.	Result.
10	Mahabook	49	M. M.	"	Opened	R
11	Goburdhone	35	H. M.	"	"	D
12	U. C.	46	H. M.	"	"	R
13	G. R.	38	H. M.	Alcohol and Dysentery	Opened and Quinine	R
14	Abba	46	M. M.	Malaria	Opened	R
15	G. C. C.	43	H. M.	Alcohol	Medicinal	D
16	Ramdass	35	H. M.	"	"	D
17	J. N. D.	40	H. M.	"	Opened (late)	D
18	Prya	37	H. M.	"	"	R
19	Ram	46	H. M.	"	"	R
20	I. B. S.	36	H. M.	"	"	R
21	T. N. B.	34	H. M.	"	"	R
22	S. N. B.	42	H. M.	"	"	R
23	B. H.	28	H. M.	"	"	D
24	W. C. B.	43	H. M.	"	"	R
25	P. N. C.	45	H. M.	"	"	R
26	Bhika	40	H. M.	Dysentery	Medicine	D
27	Ram	50	H. M.	"	"	R

28	M. N. C.	41	H. M.	Alcohol	Opened	R
29	S. B. C.	40	H. M.	Alcohol	Opened	R
30	M. N. B.	42	H. M.	"	"	R
31	P. C. B.	30	H. M.	"	"	R
32	G. C. B.	36	H. M.	"	"	R
33	Manne	37	H. M.	"	"	R
34	B. R. S.	39	H. M.	"	"	D
35	B. B. S.	42	H. M.	"	"	R
36	R. C. C.	44	H. M.	"	Medicine	D
37	R. N. D.	50	H. M.	Alcohol and Dysentery	"	D
38	J. W. C.	48	H. M.	Alcohol	Opened	D
39	K. K. D.	40	H. M.	"	"	R
40	B. M. B.	36	H. M.	"	"	R
41	N. M. B.	34	H. M.	"	"	R
42	R.	38	H. M.	"	"	R
43	B. P. D.	42	H. M.	"	"	R
44	K. N. G.	43	H. M.	"	"	R
45	S. R. D.	45	H. M.	"	"	R
46	J. M.	50	H. M.	"	"	R

No.	Name.	Age.	Caste.	Cause.	Treatment.	Result.
84	G. R. B.	38	H. M.	"	Opened.	R
85	A. K. B.	29	H. M.	"	"	R
86	Panui.	25	M. M.	"	"	R
87	D. M. B.	28	H. M.	Alcohol.	Opened	R
88	F. N. M.	30	H. M.	"	"	D
89	C. C. C.	41	H. M.	"	Opened into Stomach.	D
90	P. N. C.	34	H. M.	Dysentery.	Medicinal.	D
91	S. P. B.	35	H. M.	Alcohol.	"	D
92	S. N. R.	38	H. M.	"	Opened.	R
93	R. N. B.	43	H. M.	"	"	R
94	S. C. S.	34	H. M.	"	"	R
95	D. N. D.	30	H. M.	"	Burst Externally.	R
96	B. N. C.	29	H. M.	Dysentery.	Opened.	R
97	N. C. C.	27	H. M.	"	"	D
98	B. L. C.	28	H. M.	"	"	D
99	S. K. C.	29	H. M.	"	"	R
100	C. K. C.	44	H. M.	"	"	D

Treatment.	Number.	Death.	Per cent.
Opened	81	18	22
Medicinal	10	10	10
Burst Externally	3	1	33
„ into Lungs	3	1	33
„ into Stomach	1	1	100
„ into Bowels	2	1	50
	100	32	32

Hindu	95	
Mohomedan	5	
	—	
	100	
Age. 25 to 30	25	
31 „ 40	40	
41 „ 50	35	
	—	
	100	
Cause:—		
Alcohol	80	
Dysentery	14	
Ganja & Gooly	2	
Malaria	4	
	—	
	100	

APPENDIX II.

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